HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA

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Submitted By

DR. HARDEEP KAUR

DEPARTMENT OF OTORHINOLARYNGOLOGY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA, PUNJAB

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I hereby declare that the thesis entitled "**HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA**" submitted towards partial fulfillment of the requirements for the award of **MS in Otorhinolaryngology (ENT)** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Manish Munjal,** Professor & Head, Department of ENT, **Dr. Sanjeev Puri**, Assistant Professor, Department of ENT, **Dr. Naveen Mittal**, Professor, Department of Endocrinology. No part of this thesis has formed the basis for the award of any degree previously.

_____

DR. HARDEEP KAUR
Department of ENT,
(Otorhinolaryngology)
Dayanand Medical College & Hospital,
Ludhiana, Punjab

Date:

Place:

CERTIFICATE OF SUPERVISORS

This is to certify that the work incorporated in this thesis entitled "**HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA"** has been carried out by **Dr. Hardeep Kaur** under our direct supervision. The data incorporated in this thesis is original and genuine and has been carried out by the candidate herself.

Signature of Supervisor

Dr. Manish Munjal
Professor & Head
Department of ENT
DMC&H, Ludhiana

Signature of Co-Supervisors

Dr Sanjeev Puri
Assistant Professor
Department of ENT
DMC&H, Ludhiana

Dr. Naveen Mittal
Professor
Department of Endocrinology
DMC&H, Ludhiana

Date:

ENDORSEMENT BY THE HEAD OF THE INSTITUTE

This is to certify that the thesis entitled "**HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA "** is a bonafide research work done by **Dr. Hardeep Kaur** under the supervision of **Dr. Manish Munjal,** Professor & Head, Department of ENT, **Dr. Sanjeev Puri**, Assistant Professor, Department of ENT, **Dr. Naveen Mittal**, Professor, Department of Endocrinology, in this institution.

Principal

Dr. Sandeep Puri
Principal
DMC&H, Ludhiana

Date:

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Signature

Dr. Hardeep Kaur
Department of ENT,
(Otorhinolaryngology)
Dayanand Medical College & Hospital,
Ludhiana, Punjab

Date:

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Dedicated

to

my Family

&

Teachers

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Dr. Hardeep kaur

ABBREVIATIONS

OAE	OTOACOUSTIC EMISSIONS
DPOAE	DISTORTION PRODUCT OTOACOUSTIC EMISSIONS
TEOAE	TRANSIENT EVOKED OTOACOUSTIC EMISSIONS
PTA	PURE TONE AUDIOMETRY
OHC	OUTER HAIR CELLS
IHC	INNER HAIR CELLS
TM	TYMPANIC MEMBRANE
EAC	EXTERNAL AUDITORY CANAL
TIDM	TYPE I DIABETES MELLITUS
T2DM	TYPE 2 DIABETES MELLITUS
SNHL	SENSORINEURAL HEARING LOSS
BERA	BRIANSTEM EVOKED RESPONSE AUDIOMETRY
HBA1C	GLYCATED HAEMOGLOBIN
RBS	RANDOM BLOOD GLUCOSE
C DNA	COMPLEMENTARY DEOXYRIBONUCLEICACID
M RNA	MESSENGER RIBONUCLEIC ACID
ATP	ADENOSINE TRIPHOSPHATE
CSF	CEREBROSPINAL FLUID
STAS	SULFATE TRANSPORTER AND ANTI SIGMA FACTOR TRANSPORTER
ROS	REACTIVE OXYGEN SPECIES
EP	ENDOCOCHLEAR POTENTIAL

ABSTRACT

Introduction: OAEs are the low intensity sound produced by the outer hair cells of normal cochlea. It is simple noninvasive screening tool used to assess the function of cochlea. BERA is a non invasive test used to diagnose retro cochlear pathology**.**

Aims and Objectives-Primary Aim -To detect site of lesion (cochlear hair cell/ cochlear nerve. Secondary Aim -Correlation between diabetic profile (Blood Glucose, Serum HbA1c) and audiological parameter (PTA, OAE, BERA).

Materials and Methods: 30 diabetic patients with hearing loss who passed the inclusion and exclusion criteria selected for the study. After baseline investigations (HbA1C and blood glucose),clinical examination (external auditory canal , tympanic membrane) and detailed history, patient underwent pure tone audiometry, otoacoustic emission test and Brainstem evoked response audiometry test to detect the site of lesion. Hearing loss was correlated with diabetic profile (HbA1C and blood glucose).

Results: In majority 29 (96.7%) of patients site of lesion was outer hair cells. Hearing loss has positive correlation with HbA1c (p=0.001).Mean amplitude of otoacoustic emissions decreases with increase in HbA1C (p=0.001).Majority 21(72.4%)were males with reduced amplitude of DPOAE with respect to females 8(27.5%).As there was one diabetic patient with prolonged latency of wave V on BERA there was no correlation with increase in HbA1c .

Conclusion: In type 2 diabetic 6kHz was the frequency which was involved in maximal subjects. Male diabetic had higher incidence of involvement.

Keywords: Otoacoustic emissions, BERA, Diabetes, Sensorineural hearing loss

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INTRODUCTION

Otoacoustic emissions (OAE) are the low intensity sounds having their origin in the inner ear. These emissions are disturbed when cochlea is damaged. Otoacoustic emissions will be normal if there is retro cochlear pathology but there will be abnormal auditory brain stem responses.[1,2] Huizinga MM, Rothman et al.stated that 40.9 millions people in India are suffering from Diabetes Mellitus and by 2025 the number would be around 69.92 million.[3,4] Diabetes is a chronic health condition that causes considerable morbidity and mortality and affects the quality of life.[5] The organ of corti is the sense organ for hearing and the potential target for hyperglycemic damage that occurs in Diabetes Mellitus.

Hearing loss affects the quality of life and leads to depression, anxiety as well as social isolation [6]. Patients with diabetes mellitus often show symptoms such as dizziness, tinnitus, and hearing impairment. Dalton DS, Cruickshanks KJ Salvinelli F, Mielw A et al concluded that Diabetes Mellitus and Hearing loss (SNHL) in early studies conducted showed a weak correlation[7,8] but some later studies showed positive correlations[9].Hearing tests evaluates frequencies of 500, 1000, 2000, 3000, 4000, and 6000 Hz. The average of both ears is used for analysis.The pure tone audiometry of both ears at each frequency is obtained for frequency analysis.

According to the American National Standards Institute, hearing loss is Classified as Slight hearing loss: 16 to 25 dB,Mild hearing loss: 26 to 40 dB,Moderate hearing loss: 41 to 55 dB,Severe hearing loss: 71 to 90 dB Profound: over 90 dB.[10]

Microangiopathy in Diabetes Mellitus leads to sensorineural haring loss in diabetic patients.[11] Due to hyperglycemia deposition of glycoproteins in small blood vessels occurs that impacts the neurological function in Diabetes patient.[12,13]Amplitude and intensity of the recorded waves for otoacoustic emission is significantly lower in diabetics than in controls.[14]

Quality of life improves in diabetic patients with the treatment of hearing loss which is the chronic complication of diabetes mellitus.[15] Cochlea is damaged by the hyperglycemia that occurs in type two diabetes mellitus Due to damage of outer hair cells in cochlea hearing loss occurs in type 2 diabetes mellitus. The number of undiagnosed cases of undiagnosed diabetes across the globe is approximately 175 million currently. Cochlear hair cell status is assessed by the otoacoustic emission test battery that is simple, non invasive and efficient indicator.

On auditory stimulation sound is produced by the hair cells in the cochlea known as otoacoustic emissions or the sound which are picked up by the microphone fitted in the ear canal. Electrophysiological activity of the auditory pathway is evaluated by the brainstem auditory evoked responses. Integrity of the auditory pathway and early impairement of acoustic nerve is assessed by the this non invasive test.[16]

The outer hair cells in inner ear produce soft sounds (OAEs). These hair cells are electro motile. The main function is the active process that increase sensitivity to sound known as cochlear amplification.This sensitivity to sounds decreases due to damage and loss of outer hair cells. Sometimes without any obvious damage to outer hair cells endo cochlear potential decrease.

Helzner EP et al.[17]proposed mechanisms underlying the association between diabetes and Hearing Impairement include the combined contributions of hyperglycemia and oxidative stress to cochlear microangiopathy and auditory neuropathy. In this review, we highlight recent population-based studies of type 2 diabetes and Hearing Impairement and examine evidence for diabetes-induced pathophysiologic changes that may result in damage to the auditory system.

Thickening of the basement membrane in capillaries is implicated in the microangiopathic complications of diabetes mellitus.[18] Diabetes(T1D) leads to vascular damage and neuropathy.[19]

There are two types of Otoacoustic emissions are utilized-

(a) Spontaneous Otoacoustic emissions **–**

They are present in healthy normal hearing persons where hearing loss doesn't exceed 30 dB and may be absent in 50% of normal individuals.

(b) Evoked Otoacoustic emissions**–**

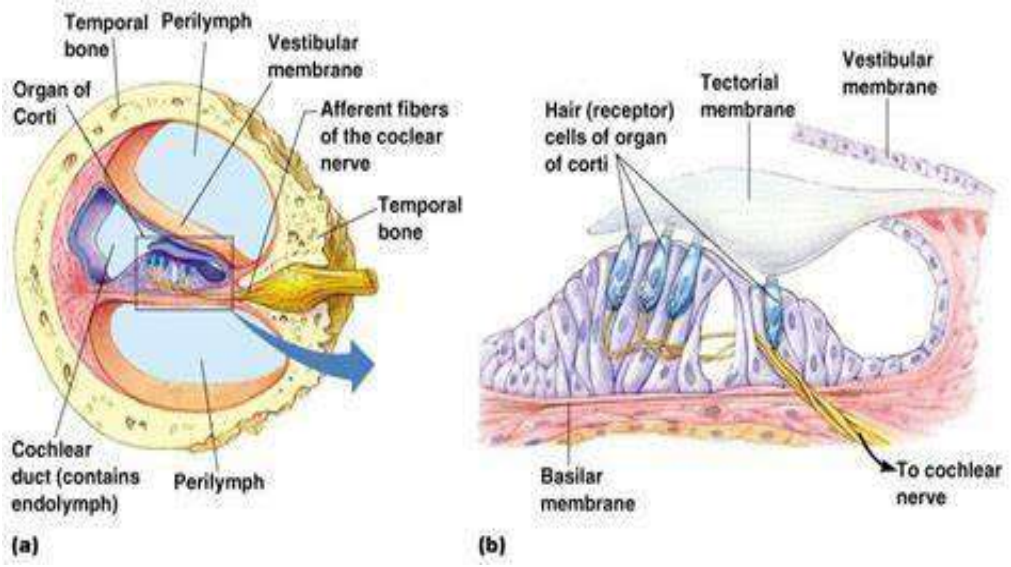
They are of two types depending on the nature of stimuli used to elicit them.

(1)- Transient evoked Otoacoustic emissions (TEOAEs) –

A series of click stimuli are presented at 80- 85 dB SPL (sound pressure level) and response recorded.

(2**)-** Distortion product Otoacoustic emissions (DPOAEs)-

Two pure tones are simultaneously presented to cochlea to produce distortion product. They are used to test hearing in range of 1000-8000Hz.



Cross section of Cochlea (16)

Practical applicability of otoacoustic emissions-

1) It is used as a screening test for hearing in presbycusis and in intellectual compromised individuals and uncooperative individuals after sedation.
2) They distinguish cochlear from retro cochlear pathology. They are also useful to diagnose retrocochlear pathology, especially auditory neuropathy incombination with BERA.Thus distinction between sensory and neural pathologies can be made.
3) This test can assess the function of outer hair cells of organ of corti which is the predominant site of injury Inototoxocity.
4) Monitor cochlear function during recovery from cochlear dysfunction.
5) OAEs can be either present or absent with 8th nerve tumours, depending on whether the cochlear blood supply has been compromised. If present, OAEs indicate possibility of hearing recovery with a conservative surgical approach.
6) OAEs are objective and require minimal patient cooperation hence can be used in malingerers.
7) This test is frequency specific and relatively consistent and stable.
8) The test is short and need not to be performed in a sound attenuated room, though a quiet ambience is essential(16)

Sound generated by the outer hair cells is transmitted by the basilar membrane to the perilymph and hence through the oval window, ossicles, tympanic membrane into the ear canal.The microphone picks up from the ear canal.[20]

AIMS & OBJECTIVES

Primary Aim:

To detect the site of lesion (cochlear hair cells/ cochlear nerve).

Secondary Aim:

Correlation between diabetic profile

- Blood Glucose
- Serum HbA1c

Audiological parameter

- PTA
- OAE
- BERA

REVIEW OF LITERATURE

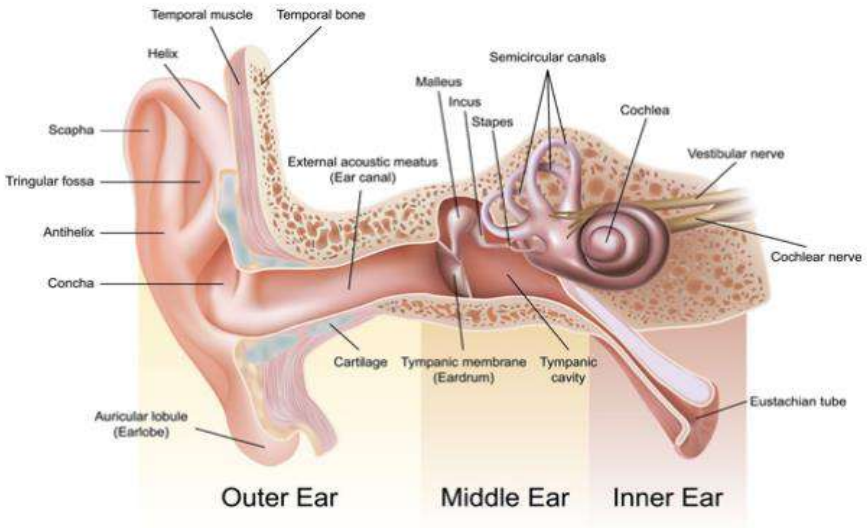
Human cochlea initially described by Eustachi in 1564 and pictorially depicted by Albini in Leyden in 1744. Neapolitan anatomist Cotugno (1761) elaborated the cochlear anatomy with corrosion casts in De aqueductibus Auris Humana Interna. Ninety years later, Corti (1851) discovered the “organ of corti” which bears his name.Otoacoustic Emissions are weak acoustic signals emitted from the cochlea. Otoacoustic Emissions are the by products of the contraction of the outer hair cells and creation of a reverse acoustic wave emitted back through the middle ear to the external ear canal. When testing these emissions, a small microphone is installed in the external ear canal to register the signals.[21]

The Otoacoustic emissions have been reviewed from the following aspect:

(i) Anatomical
(ii) Embryological
(iii) Physiological
(iv) Histological
(v) Pathological
(vi) Pathophysiological
(vii) Analytical

ANATOMICAL ASPECT

The ear is composed of three parts: the outer ear, the middle ear and the inner ear.



Anatomy of ear[22]

INNER EAR

The inner ear or the labyrinth performs the function of hearing and balance. It consists of a bony and a membranous labyrinth. The membranous labyrinth is filled with a clear fluid called endolymph while the space between membranous and bony labyrinth is filled with perilymph.

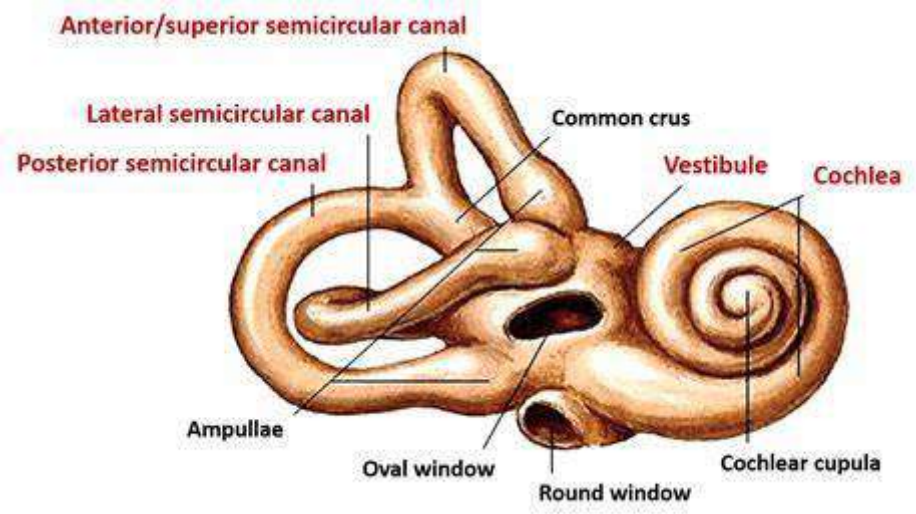
Bony Labyrinth

It consists of three parts – the vestibule, cochlea and the semicircular canals.

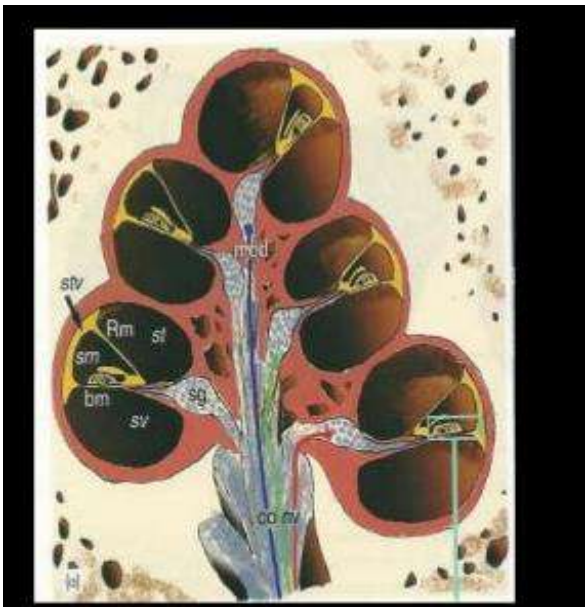
Vestibule

It is the central chamber of the labyrinth.In its lateral wall lies the oval window. The inside of its medial wall presents two recesses, a spherical recess,which lodges the saccule and the elliptical the

utricle.In the posterosuperior part of vestibule are the five openings of semicircular canals.



TheRightBony Labyrinth [23]



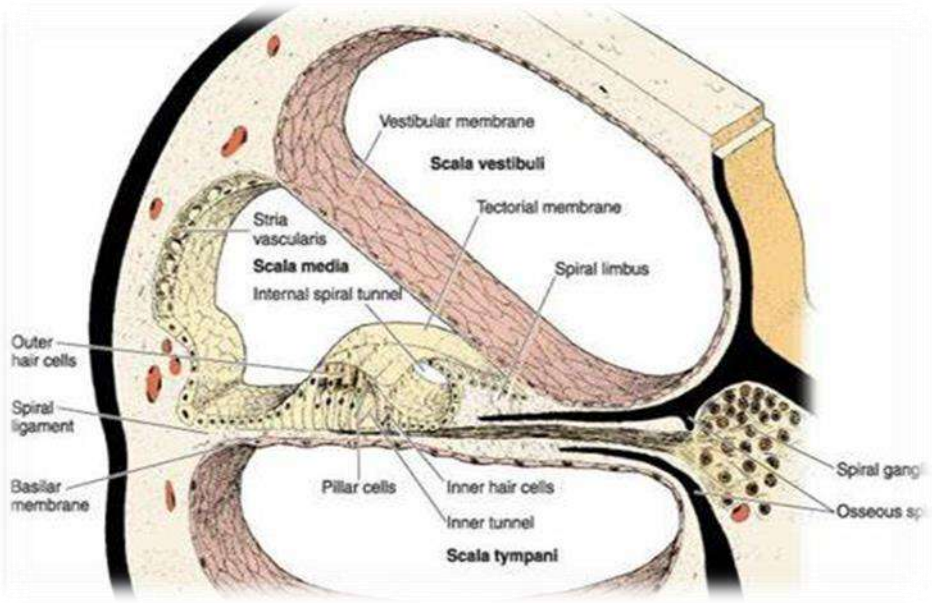
The Modiolus[24]

Semicircular canals

They are three in number,lateral, posterior and superior, and lie at right angle to each other. Each canal has an ampullated end which opens independently into vestibule and non ampullated end.The non ampullated end of posterior and superior canals unite to form the crus commune.Thus the three canals open into vestibule by five openings.

Cochlea

The bony cochlea is a coiled tube making 2.5 to 2.75 turns round a central pyramid of bone called modiolus. The osseous spiral lamina divides the bony cochlea incompletely, thus gives attachment to the basiler membrane. The bony bulge on the medial wall of middle ear , the promontory , reflects the basal coil of cochlea.



Osseous spiral lamina [25]

The bony cochlea is divided into:

(i) Scala vestibuli

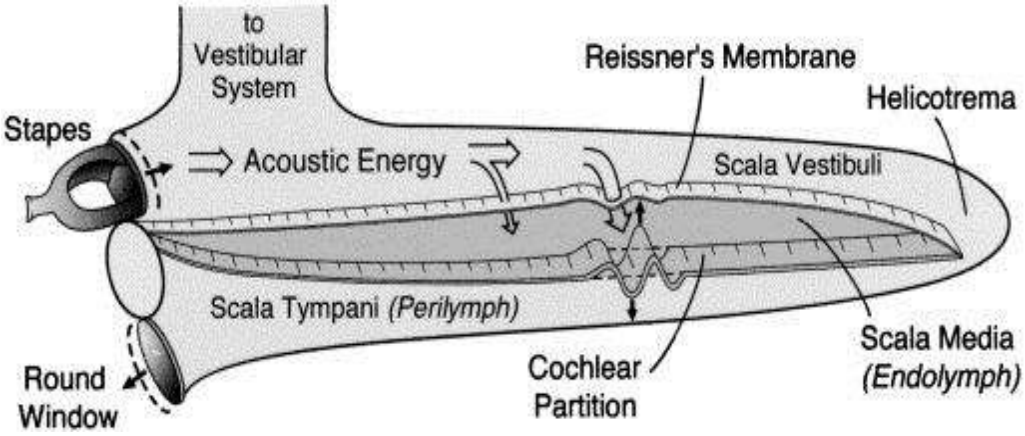
(ii) Scala media or the membranous cochlea.

(ii) Scala tympani

Perilymph:

The fluid inside the scala vestibuli and tympani communicates with the cerebro spinal fluid via the cochlear aqueduct .Scala tympani and scala vestibuli communicate with each other at the apex of cochlea through an opening, called the helicotrema .Scala vestibuli is separated by the footplate of stapes and scala tympani by the secondary tympanic

membrane from the middle ear .



Helicotrema - communication between scala tympani and scala vestibuli [26]

Membranous labyrinth

It consists of the cochlear duct, the utricle and saccule, the three semicircular ducts, and the endolymphatic duct and the sac.

Cochlear duct

It is also called scala media. Organ of Cortiresides the mechanosensory cells arranged in one row of inner hair cells (IHCs) and three to four (sometimes as many as five) rows of outer hair cells (OHCs). Bundles of sensory hairs (stereocilia) emerge from the apical poles of the sensory receptor cells. Hair cells are surrounded by several types of supporting cells, and these support cells have contact with the basement membrane. Because hair-cell stereocilia are coupled to the overlying acellular tectorial membrane, oscillations of the basement membrane cause back-and-forth deflection of the hair bundles**.** This motion is not uniform along the cochlear spiral, because the basement Membrane is narrow and rather stiff on its base but is wider and more compliant near the apex of the cochlear duct. von Bekesy (1960) was the first to show that sound sets up a travelling wave and its peak amplitude varying with respect to location on the basement membrane with frequency. High frequencies are mapped near the base while low frequencies cause maximal vibrations closer to

the apex. This leads to a tonotopical organization of the sensory cells where the location of excited hair cells along the basement membrane determines the perceived frequencies. Basement Membrane and the thin reissner's membrane subdivide the spiral canal into its three compartments. Scala Media is filled with endolymph that can be distinguished by its ion composition that is much like an intracellular character with respect to its potassium content. Scala Tympani and scala Vestibuli are filled with perilymph containing an extracellular-like ion composition and communicated at the apex of the cochlea via the helicotrema. The fluid contained within the tunnel of Corti and Nuel's spaces within the sensory epithelium resembles perilymph's composition.[27]

Reissner's Membrane

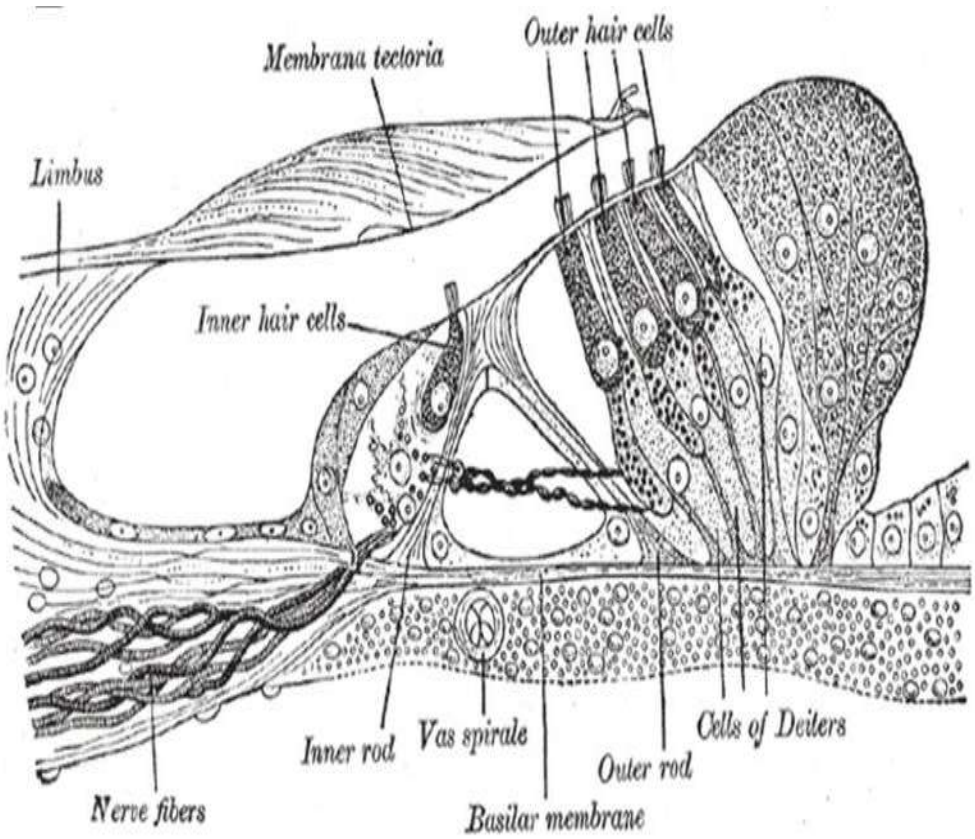
The human Reissner's membrane consists of an epithelial-cell layer facing the endolymphcompartment of the scala Media and a mesothelial facing the perilymph compartment of the scala vestibuli. These cell layers are separated by a basement membrane and form an important barrier between these two fluids that possesses completely different ion compositions. Reissner’s Membrane is involved in homoeostasis and fluid transport. Integrity of this membrane is essential for hearing to maintain the endocochlear potential (EP; +80 mV) in the cochlear duct.[28]

Basilar membrane

Basilar membrane forms the boundary between the scala media and the scala tympani and includes Claudius’ cells,Boecttcher’s cells; the organ of corti, which contains Hensen’s cells, Deiter’s cells, pillar cells, inner border cells, outer hair cells, and the inner hair cells, inner sulcus; spiral limbus, which contains the interdental cells and overlying tectorial membrane.[29]

Lateral wall

The lateral wall includes spiral Ligament,stria vascularis and the external sulcus).The spiral ligament anchors the basement membrane at the lateral aspect of the otic capsule. This attachment to the organ of Corti is characterized through the presence of tension fibroblasts that contain actin, myosin, and tropomyosin. This may generate a certain active tension that can be modulated. Besides its mechanical function, the spiral ligament plays an important role for the supply and drainage of perilymph.[30]



Organ of corti[31]

The difference between outer and inner hair cells are tabulated below [32]

Characteristic	Outer hair cells (OHC)	Inner hair cells (IHC)
Number (human)	9000 to 12,500	3000 to 3500
Arrangement of cells	Three staggered rows	A single row
Form	Cylindrical	Pear-shaped
Structure	Cytoskeleton containing motor protein (prestin, actin) that enables the cell to change its size.	Conventional cytoskeleton
Arrangement of stereocilia	Three rows of Ws	Three aligned rows
Cellular organelles	Nucleus situated at the base of the cell (basal nuclei). Abundant mitochondria aligned along the cell body and below the basal nucleus.	Nucleus situated medially in the cell. Less abundant mitochondria than in the OHC and more uniformly organized in the cell body.
Neural network	Afferent system: synapses with small endings from non-myelinated type II spiral ganglia link the cochlea to cochlear nuclei (approximately 5% of auditory nerve fibres). Approximately ten OHC per type II neuron. Efferent system: very large descending endings from both sides of the medial superior olivary complex. The endings form axo-somatic synapses with the OHC cell body.	Afferent system: synapse with large myelinated type I spiral ganglion neurons, which link the cochlea to the cochlear nuclei (95% of auditory nerve fibres). 10 to 20 type I neurons per IHC. Efferent system: arising from small neurons in the ipsilateral lateral superior olivary complex bring feedback (post-synaptic) control to the type I afferent fibre
Type of synapse	Postsynaptic cistern	Presynaptic ribbon
Neurotransmitter	Aceylcholine, GABA, neuropeptide (descending fibres)	Glutamate (ascending fibres)
Functional role	Enables local amplification of the movements of the basilar membrane. Increases the sensitivity of the internal hair cells.	Enables transduction of mechanical energy into electrical energy. Actual sensory receptors.

Tectorial Membrane

Tectorial membrane (TM) is an extracellular matrix that causes a shearing motion to stereocilia bundles when vibration enters the cochlea partition. This auxiliary mass is required to present movement to the “light weight” and therefore rather vibration-insensitive hair cells. The tympanic membrane is composed of radially running unbranched fibrils of type II and type IX collagen (type A) and highly branched fibers of type V collagen (type B) in which the thick fibers are embedded. The jelly-like matrix is composed of various glycoproteins, the tectorins and otogelin.[33]

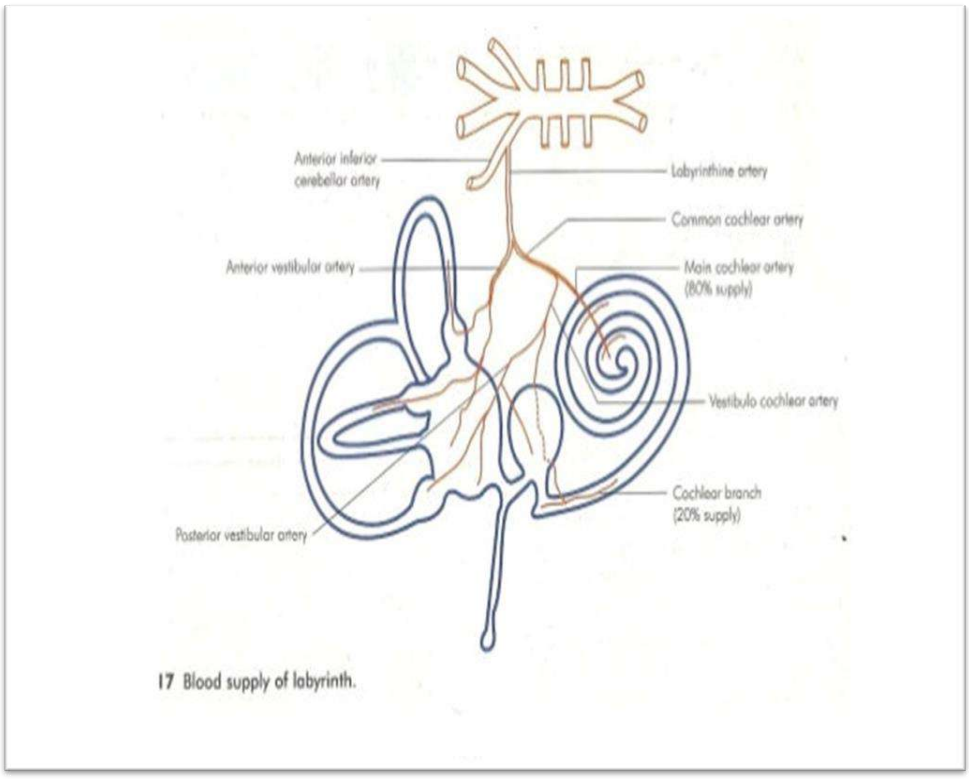
SUPPORTING CELLS

Supporting cells provide tight and buckling resistant connections of hair cells with the Basement Membrane. **Deiter cells** sustain the bases of Outer Hair Cells forming a “seat” for the basal portions of these sensory cells and send a phalangeal process to the reticular lamina completing their structural support. **Hensen, Claudius, and Boettcher cells** shape the lateral segment of the organ of Corti.[34]

HUMAN SPIRAL GANGLION

The human cochlea from normal hearing individuals contains approximately 35,000 afferent neurons that are bipolar with soma located in the helical Rosenthal's canal (in the modiolus along the 1¾ turns of the cochlea. The spiral ganglion terminates in a bulge containing the cell bodies of neurons innervating hair cells of the third turn. Peripheral processes run within a bilamellar bony space of the osseous spiral lamina to the habenula perforata to exit this bony canal and innervate the hair cells.[34]

BLOOD SUPPLY OF COCHLEA



Blood supply of cochlea [35]

EMBRYOLOGICAL ASPECT

The Chronological embryological development of the human cochlea is tabulated below.[36]

Embryological age	Development of inner ear
4 – 5 weeks	Otic vesicle (Dorsal and ventral pouch), endolymphatic sac (dorsal pouch) and vestibular structure formation occurs from ectodermal cells
10 weeks	Cochlea forms two and half turn coil
18- 20 weeks	Complete development of cochlea occurs from ventral pouch of otic vesicle

PHYSIOLOGICAL ASPECT

TRANSDUCTION OF MECHANICAL ENERGY TO ELECTRICAL IMPULSES

Movements of the stapes footplate which are transmitted to the cochlear fluids, move the basilar membrane and set up a shearing force between the tectorial membrane and the hair cells. The distortion of hair cells is the origin of cochlear microphonics, which initiates a nerve impulse. A sound wave, depending on its frequency, attains maximum amplitude at a particular site on the basilar membrane and stimulates that very segment. Higher frequencies are represented in the basal turn of the cochlea and the progressively lower ones towards the apex.[37]

NEURAL PATHWAYS

Hair cells get innervation from the bipolar cells of spiral ganglion. Central axons of these cells collect to form the cochlear nerve which goes to the ventral and dorsal cochlear nuclei. From there, both crossed and uncrossed fibres travel to the superior olivary nucleus, lateral lemniscus, inferior colliculus, medial geniculate body and finally reach the auditory cortex of the temporal lobe.[37]

ELECTRICAL POTENTIALS OF COCHLEA AND CRANIAL NERVE VIII

Four types of potentials have been recorded; three from the cochlea and one from cranial nerve VIII fibers.

1. Endocochlear potential
2. Cochlear microphonic
3. Summating potential from cochlea
4. Compound action potential from nerve fibres

Endocochlear potential

It is a direct current potential recorded from scala media. It is +80 mV and is generated from the stria vascularis by Na+/K+-ATPase pump and provides source of energy for cochlear transduction. It is present at rest and does not require sound stimulus. This potential provides a sort of “battery” to drive the current through hair cells when they move in response to a sound stimulus.

Cochlear microphonic

When basilar membrane moves in response to sound stimulus, electrical resistance at the tips of hair cells changes allowing flow of K+ through hair cells and produces voltage fluctuations called cochlear microphonic. It is an alternating current It is an alternating current potential.[38]

Summating potential

It is a Direct Current potential and follows “envelope” of stimulating sound. It is produced by hair cells. It may be negative or positive.

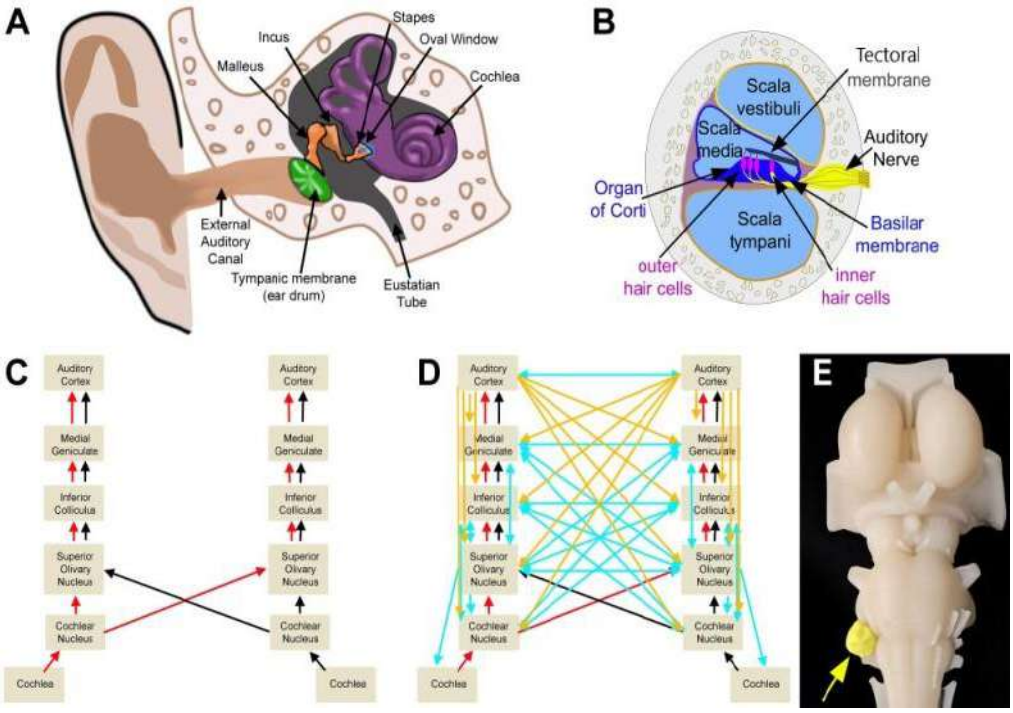
Summating Potential has been used in diagnosis of Ménière's disease. It is superimposed on VIII nerve action potential. Both Cochlear Microphonics and summating potential are receptor potentials as seen in other sensory end-organs. They differ from action potentials in that: (i) they are graded rather than all or none phenomenon, (ii) have no latency, (iii) are not propagated and (iv)have no postresponse refractory period.[38]

Compound action potential

It is an all or none response of auditory nerve fibres.

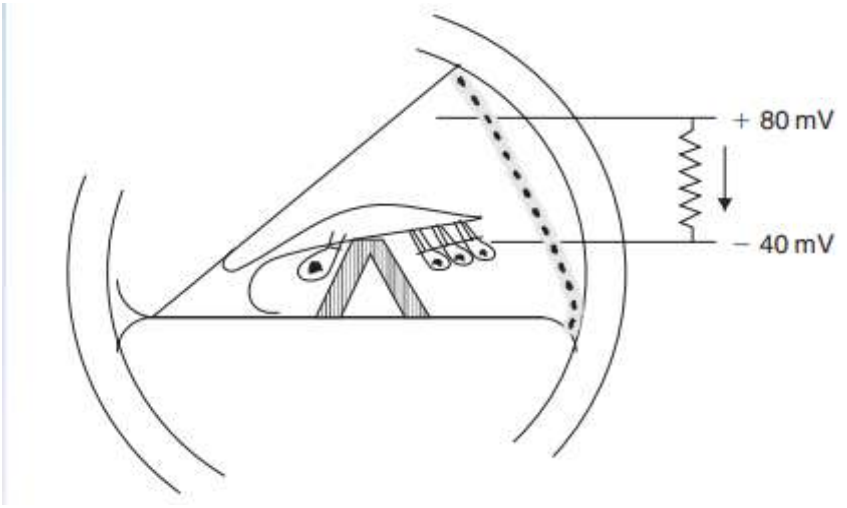
VESTIBULAR NERVE

Vestibular or Scarpa's ganglion is situated in the lateral part of the internal acoustic meatus. It contains bipolar cells. The distal processes of bipolar cells innervate the sensory. epithelium of the labyrinth while its central processes aggregate to form the vestibular nerve.



Auditory Circuits. A. Outer, middle, and inner ear. B. Cross section of the cochlea. C. Ascending auditory pathway. D. Ascending (red/black), descending (cortical: orange; brain-stem: blue), and crossed (blue) auditory circuits. E. Model representation of an acoustic neuroma.[39]

CENTRAL VESTIBULAR CONNECTION



Davis battery model of transduction [38]

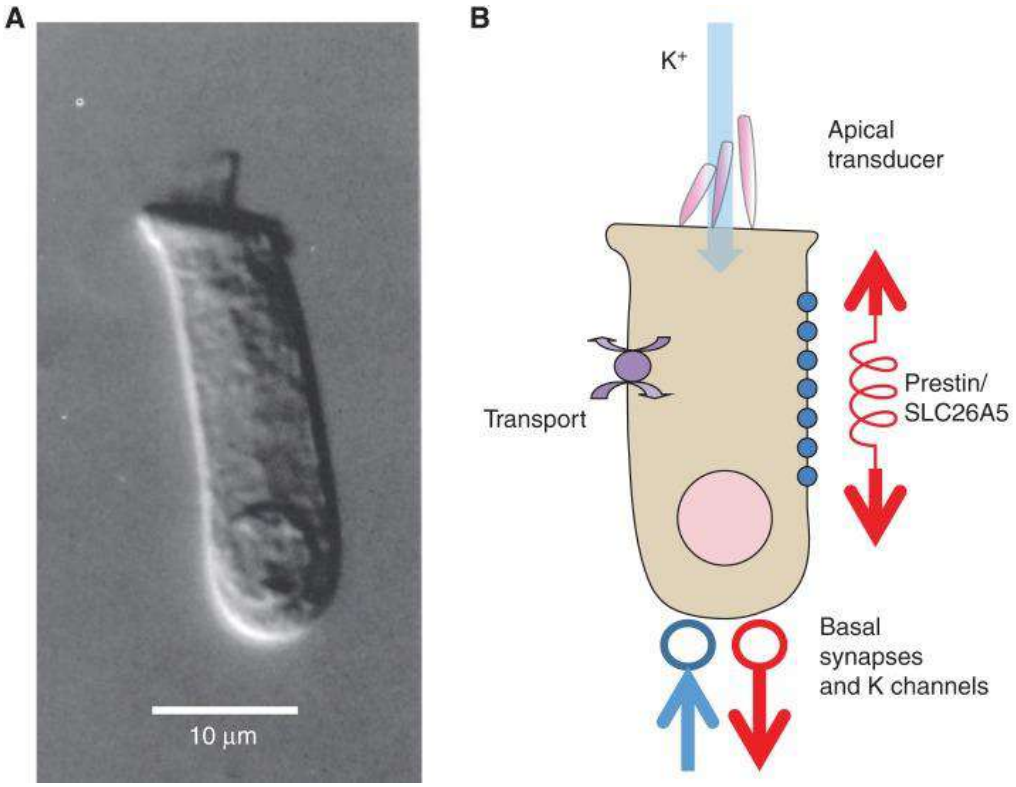
INNER EAR FLUIDS AND THEIR CIRCULATION

Perilymph resembles extracellular fluid and is rich in Na ions. It fills the space between the bony and the membranous labyrinth. It communicates with CSF through the aqueduct of cochlea which opens into the scala tympani near the round window. In fact this duct is not a direct communication but contains connective tissue resembling arachnoid through which perilymph percolates. There are two views regarding the formation of perilymph: (i) It is a filtrate of blood serum and is formed by capillaries of the spiral ligament and (ii) it is a direct continuation of cerebrospinal fluid and reaches the labyrinth via aqueduct of cochlea.[40]

Endolymph fills the entire membranous labyrinth and resembles intracellular fluid, being rich in K ions. It is secreted by the secretory cells of the stria vascularis of the cochlea and by the dark cells (present in the utricle and also near the ampullated ends of semicircular ducts). There are two views regarding its flow: (i) longitudinal, i.e. endolymph from the cochlea reaches saccule, utricle and endolymphatic duct and gets absorbed through endolymphatic sac, which lies in the subdural space and (ii) radial, i.e. endolymph is secreted by stria vascularis and also gets absorbed by the stria vascularis.[40]

PHYSIOLOGY OF OUTER HAIR CELLS

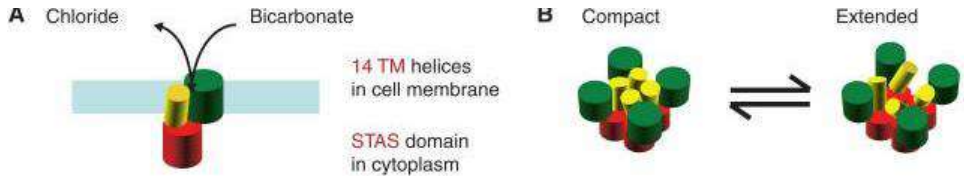
The group of cells that are responsible for hearing are the outer hair cells located in the inner ear. These hair cells are sensitive to low sound pressure levels. This property of outer hair cells is defined as the process of “cochlear amplification”. Failure of function of and loss of outer hair cells leads to hearing loss in old age or due to chronological medical conditions such as diabetes mellitus.[41]



The elements of the mammalian outer hair cell (OHC). (A) An isolated OHC from turn 2 (~5–10 kHz region) of the guinea pig cochlea. The apical stereocilia are apparent at the apical surface. (B) Schematic OHC showing the location of prestin/SLC26A5 down the basolateral surface, generating longitudinal forces. Transport to regulate pH_i is presumed to be collocated as a parallel property of prestin. K^+ ions from scala media enter through the mechanoelectric transducer channels at the apex and exit through the basally located K^+ channels. Both afferent (red) and efferent (blue) terminals are located at the base of the cell.[41]

Mammalian outer hair cells possess a very characteristic V-shaped bundle of stereocilia, serving to inject current into the cells when deflected. As sensors of the movement of the basilar membrane, outer hair cells thus form part of a local mechanical-electrical-mechanical feedback loop to control basilar membrane tuning.

There are several constraining observations: (1) The lateral membrane of the outer hair cells is packed with a particle about 8 nm in diameter; (2) A change in the outer hair cells membrane potential is accompanied by a gating charge movement, or equivalently the cell membrane capacitance is voltage dependent; (3) The charge movement is blocked by the amphiphilic anion salicylate (aspirin, with an additional methyl group has the same effect); and (4) outer hair cells only acquire motile properties progressively during a short period of development.The identification of prestin by using a subtracted complementary DNA (cDNA) library prepared from the messenger RNA (mRNA) of isolated hair cells in principle solves most of these problems.[42]



Models for prestin. (A**) Organization of prestin/SLC26A5 in the membrane as a monomer. Two mobile components of the protein are placed in the membrane whereas the carboxy-terminal region, containing the sulfate transporter and anti-sigma factor antagonist (STAS) domain is cytoplasmic . (**B**) Hypothetical model for coassembly into a tetramer. The carboxy-terminal region (red) forms a base against which the oligomer can deform, rapidly shifting between a compact (cell depolarized) and an extended configuration (cell hyperpolarized).**

PATHOLOGICAL ASPECT

Effect of hypertension on cochlea

Kirbac A, Boke B et al study on effects of primary arterial hypertension on cochlear function included 34 patients with primary hypertension and 17 healthy adults. The Cochlear function was assessed with conventional audiometry (0.125-8 kHz), ultra-high frequency audiometry (10-16 kHz), the transient evoked otoacoustic emission (TEOAE) test, and the distortion product otoacoustic emission (DPOAE) test. Hearing thresholds at 8, 10, 12.5, 14, and 16 kHz were significantly poorer in the hypertensive group than in the control group . There was no significant difference in the mean conventional thresholds between the groups. Compared to the control group, the patient group exhibited statistically significant lower amplitudes of TEOAE and DPOAE.[43]

Effect of renal disease on cochlea

Bergstrom L,Thompson et al study on renal disease with respect to pathology, treatment, and effects on the ear included twenty-six hemodialysis and renal transplant patients who were studied for the effects of age, duration of kidney failure, amounts and types of treatment, disturbed calcium metabolism, and vascular disease on clinical and pathologic inner ear manifestations.

Three subgroups of patients were compared with each other: (1) Those having hearing loss of unknown etiology, (2) Those having strial deposits, and (3) Those having neither, severity of treatment was not necessarily predictive for severity of inner ear disease. Within each subgroup there was wide variability and almost complete overlap for the factors studied when the three groups were compared. Cochlear strial deposits seem to bear little relationship to disturbed calcium meatbolism or to vascular disease, but their size may be related to the presence of hearing loss.[44]

Effect of syphilis on cochlea

Hızlı Ö, Kaya S, Hızlı P et al. study on Stria vascularis and changes in the cochlear hair cells in syphilis included 13 human temporal bone samples from 8 patients with syphilis (syphilis group), as well as 12 histopathologically normal samples from 9 age-matched patients without syphilis (control group) comparison was made between the two groups, the mean area of the stria vascularis (measured with conventional light microscopy connected to a personal computer) and the mean percentage of cochlear hair cell loss . It was concluded that syphilis led either to complete loss of the organ of Corti or to significant loss of cochlear hair cells. But the area of the stria vascularis did not show any change.[45]

Effect of chronic suppurative otitis media on cochlea

Cureoglu S, Schachern PA et al retrospective study on cochlear changes in chronic otitis media was conducted on fifteen temporal bones with unilateral chronic otitis media and compared with contralateral normal temporal bones. The loss of outer and inner hair cells was common in the basal turn of the cochlea in temporal bones with chronic otitis media compared with control ears. There was no difference in the number of spiral ganglion cells in the chronic otitis media and contralateral ears. The areas of stria vascularis and spiral ligament in the basal turn decreased significantly in the ears with chronic otitis media compared with control ears. There were no significant differences between the ears with chronic otitis media and the contralateral ears for any of the regions characterized by the presence of types I-IV fibrocytes.[46]

Effect of noise on cochlea

In the Le, T.N., Straatman, L.V., Lea, J. et al study on noise induced hearing loss, degeneration of auditory nerve fibres along with loss of outer hair cells both was seen in temporal bone histopathology in a mouse model while damage of inner hair cells was limited. Destruction of the organ of Corti is consequent to two mechanisms:

mechanical destruction by short exposure to extreme noise intensity or metabolic decompensation after noise exposure over a longer period of time. Noise intensities levels above 130 dB sound pressure causes to mechanical destruction of organ of corti, disruption of cell junction and mixing of endolymph with perilymph. The pathology observed as a result of metabolic decompensation includes stereocilia disruption, swollen nuclei, swollen mitochondria, cytoplasmic vesiculation, and vacuolization. Current theories of metabolic damage center on the formation of free radicals or reactive oxygen species (ROS) reactive oxygen species emerge immediately after noise exposure and persist for 7–10 days thereafter, spreading apically from the basal end of the organ of Corti, thus widening the area of necrosis and apoptosis.[47]

Effect of diabetes on cochlea

Akinpelu OV, Mujica-Mota M, Daniel SJ et al.analysed the effects of type 2 diabetes mellitus on hearing function. They concluded that the incidence of hearing loss ranged between 44% and 69.7% for type 2 diabetics, significantly higher than in controls. The mean pure tone audiometric thresholds were greater in diabetics than in controls and Auditory brainstem response (ABR) wave V latencies were also statistically significantly longer in diabetics when compared to control groups.[48]

Hao J, Fu X, Zhang C et al conducted a study on early detection of hearing impairment in patients with diabetes mellitus utilizing otoacoustic emissions. The study concluded that The distortion product otoacoustic emission (DPOAE) amplitudes in diabetics were significantly lower than that in controls.[49]

Hou Y, Xiao X, Ren J et al study on auditory impairment in young Type 1 Diabetics concluded that Type 1 diabetics exerted higher auditory threshold, slower auditory conduction time and cochlear impairment.[50]

Cho WK, Kang WS et al interpreted auditory brainstem evoked responses and distortion product otoacoustic emissions in diabetic

patients with normal hearing. 45 ears in patients with type 2 diabetes mellitus and 85 ears in non-diabetic patients. The study concluded that Diabetic subjects showed significantly more prolonged absolute peak latencies (I, III, V) and inter-peak latencies (I-V, III-V) than non-diabetic subjects.[51]

Spankovich C, Long GR et al DPOAE study in young adults with Type-1 Diabetes revealed a reduced cochlear function.[52]

Botelho CT, Carvalho SA et al study in young patients with type 1 diabetes concluded that abnormal DPOAEs responses were found more frequently than the alterations in TEOAEs and pure-tone audiometry, suggesting that DPOAEs evaluation is the most sensitive and it could be used for monitoring the progression of cochlear damage during the early stages of hearing impairment.[53]

Di Nardo W et al study concluded that IDDM patients show an early abnormality of the micromechanical properties of the outer hair cells and damage is limited to the higher frequencies and can be detected only by DPOAEs.[54]

Lisowska G, Namysłowski G, Morawski et al analysis concluded that the mean amplitudes of various DPOAEs were significantly reduced in the diabetic group compared with control subjects. ABR latencies were longer in diabetic patients when compared with those of control subjects.[55]

Ottaviani F, Dozio N, Neglia CB, Riccio S et al otoacoustic emission study concluded that the mean intensity of the response was lower in diabetic subjects than in controls.[56]

Akcay G, Danısman B, Basaranlar G et al study concluded significant decrease was also found in distortion product otoacoustic emission amplitudes at 4, 6, 8, and 10 kHz in the high blood glucose and diabetes mellitus groups compared to the control group.[57]

Felício, J.S., de Souza. et al. study concluded that abnormal otoacoustic emissions responses were noted in patients with diabetes.[58]

Horikawa C, Kodama S, Tanaka S et al analysis noted a higher prevalence of hearing impairment in diabetic patients compared with nondiabetic patients which was consistent regardless of age.[59]

Fukushima H, Cureoglu S, Schachern PA, Paparella MM, Harada T, Oktay MF et al study concluded that walls of the vessels of the basilar membrane and stria vascularis was thicker and loss of cochlear hair cells was noted in insulin taking group than in controls.[60]

PATHOPHYSIOLOGICAL ASPECT

Glucose is the primarysource of energy for the cochlea. The regulation of glucose uptake and metabolism is one of the major actions of insulin. High-affinity insulin receptors exist in the cochlea, but the role of insulin within the inner ear is not believed to regulate glucose uptake. This energy is required to maintain the large voltage gradient between the sensory receptors of the inner ear and the cochlear fluids, called the endocochlear potential. Experiments creating hypoglycemia reduce the endocochlear potential and cochlear microphonic, a receptor potential primarily geneated by the outer hair cells (OHCs). The reduction in DPOAEs observed by Zuma e Maia and Lavinsky was primarily limited to the more metabolically demanding basal portion of the cochlea approximately 60 minutes after induction of hypoglycemia.[61]

Biochemical Hypotheses

Diabetes mellitus, or more specifically hyperglycemia, initiates a complex cascade of biochemical consequences. Three main effects are nonenzymatic glycation, activation of the polyol pathway, and generation of reactive oxygen/nitrogen species. Metabolic processes disrupted include energy production, abnormal accumulation of metabolic by-products, nitric oxide and glutathione dysregulation, glycation (advanced glycation end products), lipid balance abnormalities, and protein synthesis dysfunction. Tissue damage associated with diabetes mellitus includes endothelial, neural, extracellular, and collagen compromise. Upregulation of vascular endothelial growth factor and nitric oxide isoforms has been demonstrated in the cochlea of patients.

Increased oxidative stress also has been implicated in diabetes mellitus pathogenesis and comorbidities associated with diabetes mellitus including hearing loss. Attempts have been made to correlate oxidative stress with glycated hemoglobin (Hb_{A1c}). Autoimmune factors have been associated with both diabetes mellitus and hearing

loss. However, the precise interaction of autoimmune disease, diabetes mellitus, and hearing loss has been elusive.[61]

Noise and Diabetes Mellitus Interaction

Noise-induced damage is related to both mechanical and metabolic compromise. Overexposure to noise can alter cochlear homeostasis resulting in excessive reactive oxygen/nitrogen species, vascular changes (an initial ischemia followed by reperfusion), activation of apoptosis-like pathways (e.g., Bcl-2 family), excitotoxic events (excessive glutamate), and subsequent cellular damage. The study concluded that more significant loss of outer hair cells in noise-exposed patients with diabetes mellitus compared with noise-exposed controls but without consideration of molecular mechanisms and relationship with glucose metabolism. McQueen et al found significant basement membrane thickening of the cochlea (microangiopathy) in patients with diabetes mellitus, however only in the combination with obesity and/or exposure to noise. Fujita et al showed significant changes to spiral ganglion despite preservation of hair cells following noise exposure in diabetes mellitus compared with controls.[61]

Age-Related Hearing Loss and Diabetes Mellitus

Vasilyeva et al examined the interaction of age and DM in two animal models, one representative for Type 1diabetes mellitus and a second representative of Type 2 diabetes mellitus. The results concluded reduced auditory function in both models, but the ABR thresholds and wave I amplitudes were significantly altered only in the Type 2 diabetes mellitus model.

Ototoxicity and Diabetes Mellitus

Garcia-Quiroga et al demonstrated protection against kanamycin-induced ototoxicity in hyperglycemic animals. Kanamycin competes with glucose transport and thus, paradoxically hyperglycemia may help control this pathway for damage.[61]

ANALYTICAL ASPECT

OTOACOUSTIC EMISSIONS

The sound produced by the outer hair cells of cochlea transmitted to external auditory canal via middle ear. Inner ear status can be assessed by the otoacoustic emission screening test.[62]

SPONTANEOUS OTOACOUSTIC EMISSIONS

The otoacoustic emissions that are present even without external stimuli and can be measured with microphone placed in external auditory canal.It is perceived as tinnitus in 1-9% patients.Their frequencies are between 500 Hz to 4,500 Hz. These occurs even in the absence of a stimulus, but they only occur in less than one half to 60% of normal hearing individuals (63).

TRANSIENT OTOACOUSTIC EMISSIONS (TOAE)

Cochlear non linearity and Outer hair function can be assessed by Transient Otoacoustic Emission test. For assessing the cochlear function acoustic data is acquired in battery including TOAEs'.(64)Click and tone burst stimuli are used in TEOAE's test. Multiple nerve fibres can be evoked by abrupt onset, slow duration and frequency range upto 4 kHz click stimuli.[65] In contrary to obtain more specific response tone burst stimuli are given at narrow frequency range especially at lower frequencies.[66]Multiple stimuli are required and averaged .Response emissions are recorded at 2 to 23-millisecond latencies corresponding to the frequency of the stimulus. Lower frequencies travel further towards the cochlear apex and require a longer latency whereas Higher frequencies propagate a shorter distance along the basilar membrane to the base and need a shorter latency.[67]

DISTORTION PRODUCT OTOACOUSTIC EMISSIONS(DPOAE)

Two simultaneous pure tone stimuli (f1-f2) are given in this procedure. For detecting high frequency hearing loss DPOAE's is the most sensitive screening test modality which can be customized to assess

frequencies that match the patient's audiogram.[68] When measuring DPOAE, the frequency calculated from the formula: 2f1-f2. Frequency ratio (f2/f1) of 1.2 provides the best accuracy in separating normal hearing patients from those with hearing loss.[69] In normal hearing people and healthy cochlea OAE will be present. Mild hearing impairment can be assessed by the OAE test. Damage to the outer hair cells from noise trauma or ototoxic medications can appear on OAE before presenting on an audiogram. If there is some dyfunction of cochlea OAE will be absent.[70]

MATERIALS AND METHODS

All Adult diabetic patients with hearing loss presenting in the ENT and endocrinology clinic of Dayanand Medical College and Hospital Ludhiana, between March 1st, 2021 to August 31st, 2022 were included in this prospective study.

INCLUSION CRITERIA

Adult diabetic patients with hearing impairment were included in the study after informed consent.

EXCLUSION CRITERIA

1. Patients with hearing loss secondary to trauma (Head injury) with documented temporal bone fracture.
2) Presence of any external or middle ear pathology (e.g. stenosis of EAC, otitis media, tympanic membrane perforation, cyst, otosclerosis).
3) Family history of sensory neural hearing loss.
4) Noise exposure.
5) Patients with Ear infections
6) Neoplastic lesions of temporal bone.
7) Patients who not consenting for the study.

METHODOLOGY

Careful selection of subjects was done after applying all the inclusion and exclusion criteria and informed consent was taken.30 patients who passed the criteria were selected for the study.

RESEARCH DESIGN

The study was an observational study conducted on adult Diabetic patients with hearing loss.It was a prospective study to detect the site of lesion by means of audiometric evaluation (Pure Tone Audiometry, Otoacoustic Emissions,BERA)and correlated with the Diabetic profile (blood glucose,HBA1C). A detailed history was taken to enquire about complaints of hearing loss which was analysed prospectively regarding-

(a) Duration of the disease

(b) Blood glucose levels

(c) Serum HbA1c.

CONSENT

An informed consent was obtained from the patient explaining the need of monitoring in diabetic patients with hearing loss explaining the need to rule out the site of lesion. Patients were motivated to remain on regular follow up.

HISTORY

A detailed history was taken to enquire about diabetic patients with complaints of hearing loss and was analysed prospectively regarding Duration of the disease, blood glucose levels, serum HbA1c, Treatment modalities (OHA, insulin) after thorough ENT examination- Examination of the Ear, external appearance Otoscopic examination.

Otoscopic examination was undertaken with Welch and Allyn hand held clinical otoscope to detect any external ear or Tympanic membrane pathology. Pure Tone Audimetry,Otoacoustic Emission Test and BERA was performed to detect site of lesion.

PATIENTS

AGE

The average age of the group was between 31- 50 yrs with minimum age being 19 yrs and maximum age being 70 yrs of age group.

SEX

09 females and 21 males were selected for inclusion in the study.

AUDIOMETRIC TESTING

(a) Baseline PTA

(b) Otoacoustic Emissions

(c) BERA were conducted in all selected subjects.

TEST ENVIRONMENT

Pure tone Audiometry was performed in standard sound treated two room setup, with noise limits within permissible limits according to Standard for Maximum Permissible Ambient Noise levels. DPOAE and BERA recordings were done in sound treated room with noise levels within permissible limits according to Standard for Maximum Permissible Ambient Noise levels.

MEASUREMENT OF PTA THRESHOLDS

Pure tone Audiometry was conducted using commercial audiometer ARPI 2009 and audiometric headphones Telephonic TDH39 with tone stimulus ranging from 0.25 to 8 kHZ (.25 ,.5 ,1,2,4 and 8 kHZ) and BC thresholds with bone vibrator B-70 with tone stimulus ranging from 0.25 to 4 kHZ (.25 ,.5 ,1,2 and 4 kHZ) using the standard modified Hughson -Westlake technique.The response was labelled as hearing loss according to WHO classification (1980).

Degree of Hearing loss-

Mild Hearing loss – 26 - 40 dBHL

Moderate Hearing loss – 41 -55 dBHL

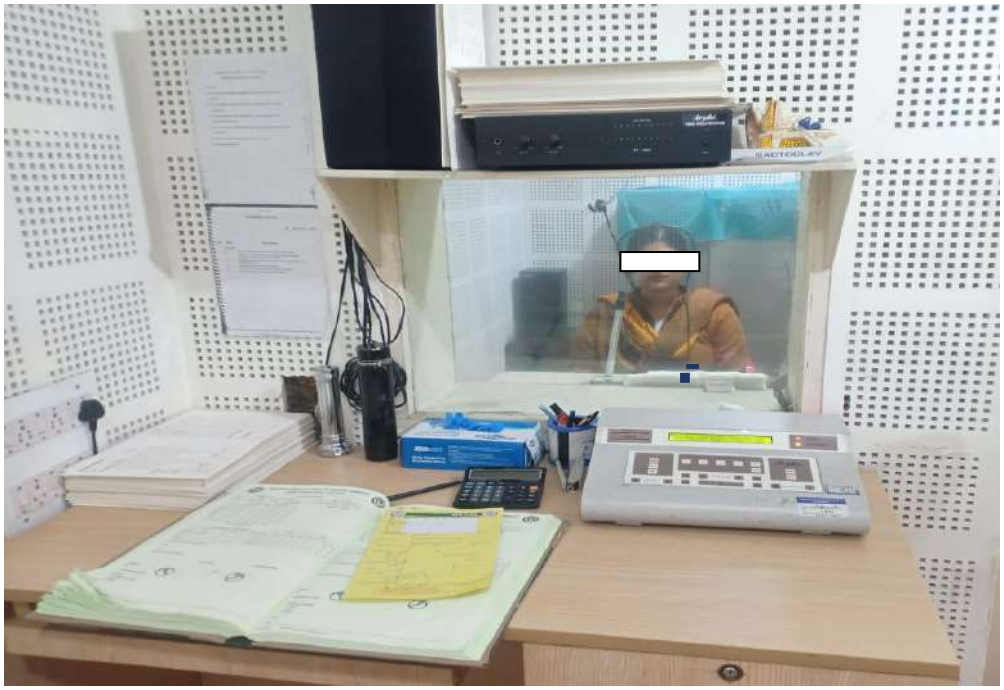
Moderately severe 56-70 dBHL

Severe 71 – 90 dBHL

Profound More than 90 dBHL

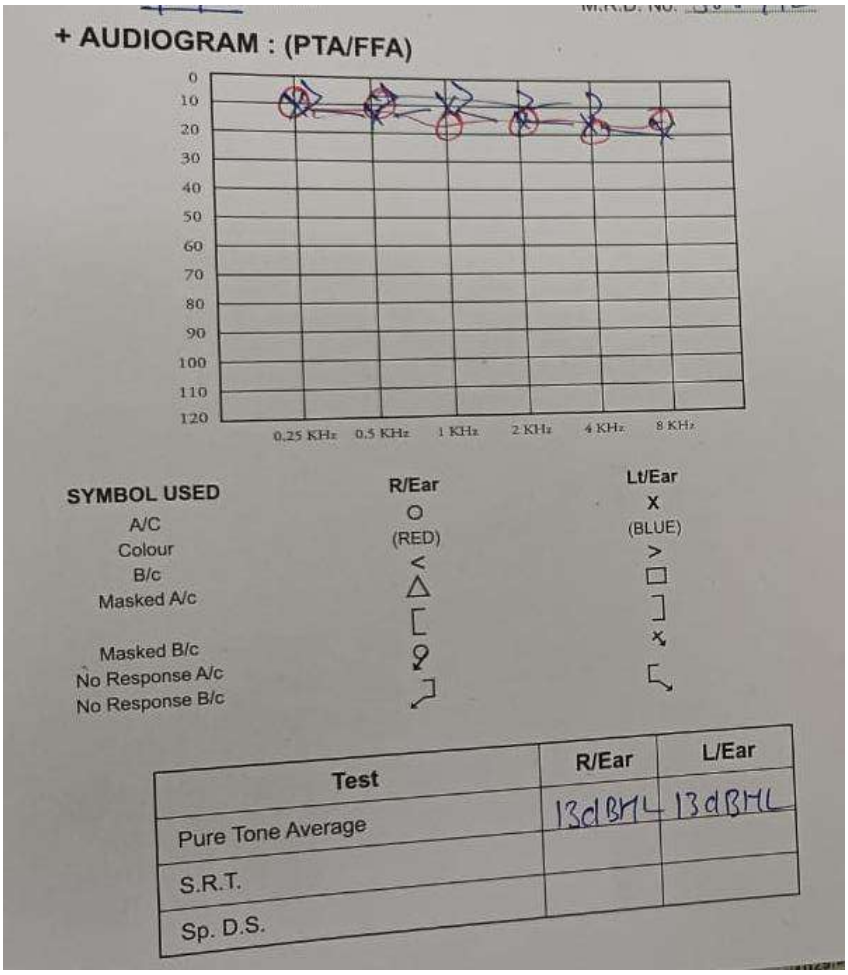
Total Hearing loss

The hearing thresholds on audiometry were measured in both ears separately.The findings were expressed as mean of right and left ear.

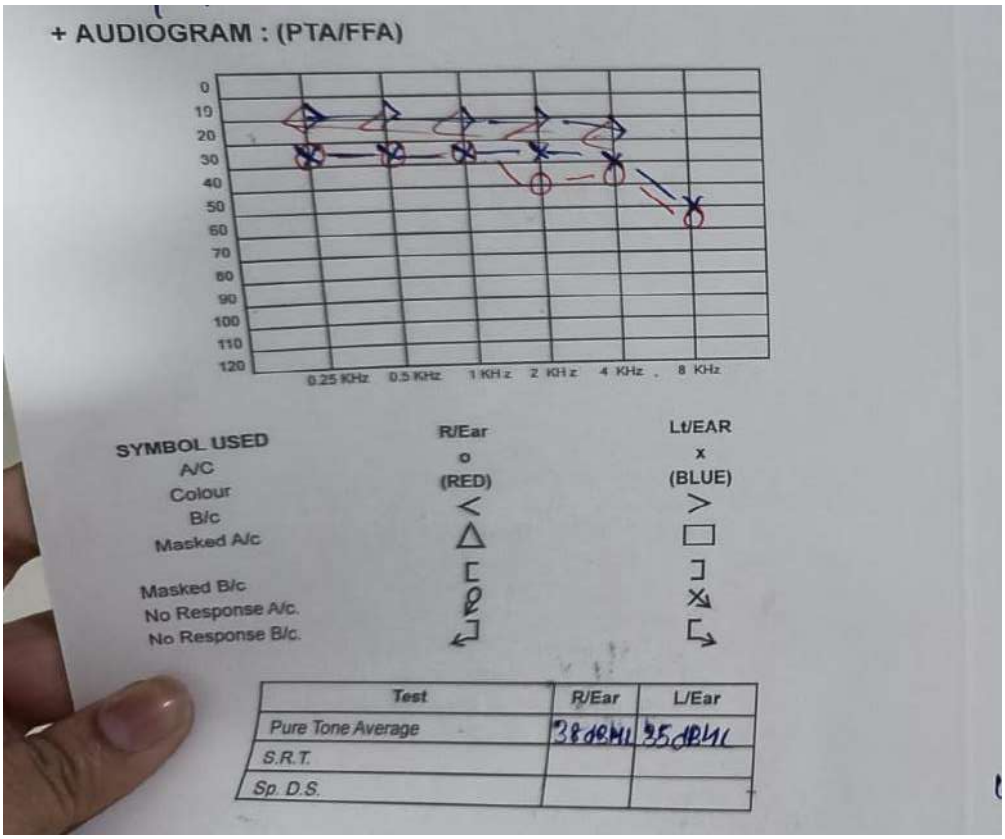


STANDARD TWO ROOM SETUP FOR PTA WITH AUDIOMETER

PURE TONE AUDIOMETER



NORMAL PTA GRAPH



PURE TONE AVERAGE GRAPH SHOWIING SENSORINEURAL HEARING LOSS

MEASUREMENT OF DPOAEs

DPOAEs were recorded using commercial NEUROAUDIO OAE equipment using NEUROSOFT 7 version, according to DP Gram procedure. A snugly fitting probe containing a sound source and a microphone was inserted in the EAC.

- Prerequisite for obtaining OAEs are-
- Unobstructed EAC
- Seal of the ear canal probe
- Optimum positioning of the probe
- A quiescent patient
- A relatively quiet recording ambience.

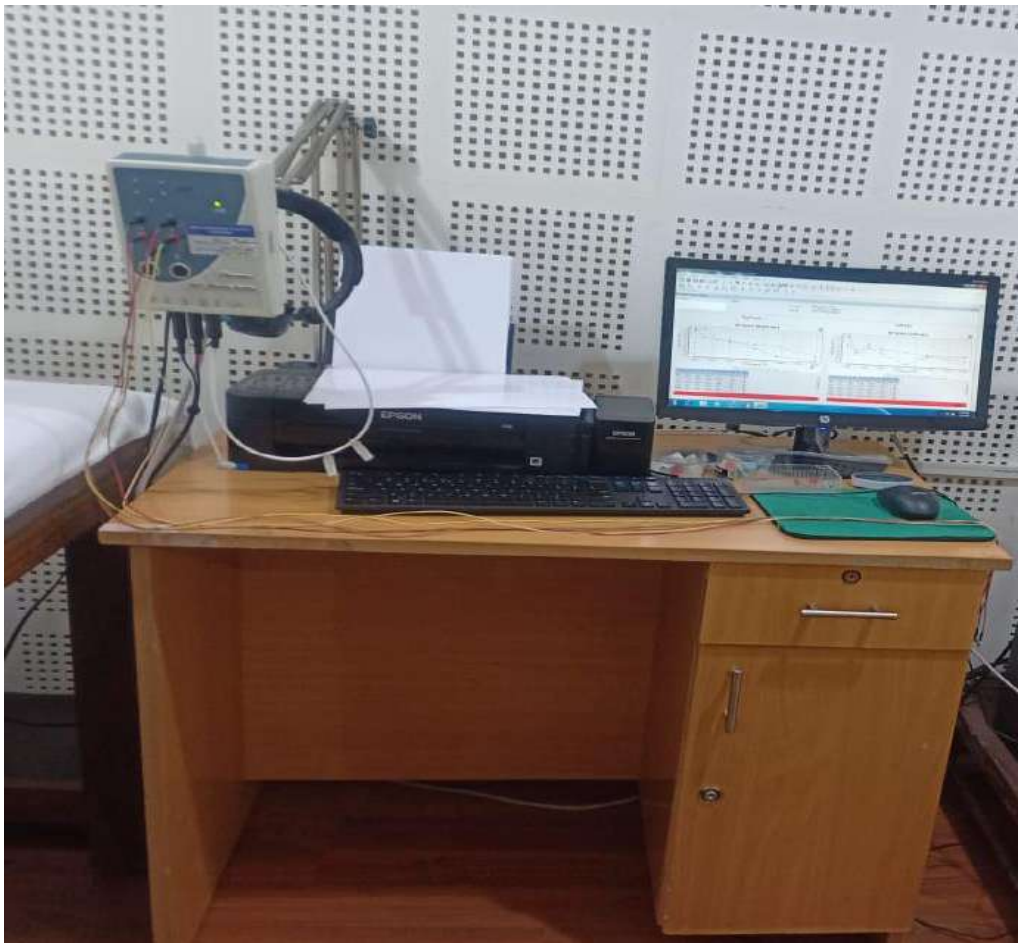
DPOAEs were measured by presenting two simultaneous presentation pure tone stimuli, conventionally with frequencies f1 and f2 (in Hz) and at intensity levels designated as L1 and L2.(in dB)

Conventionally f1 >f2 with f2 corresponding to the audiometric frequencies of 1,2,3,4,6 and 8 kHZ.The DPOAEs were considered indicative of cochlear damage and BERA indicative of retrocochlear pathology. As for Audiometry, the evoked otoacoustic emissions were measured independently in each ear. Comparisons were made between the baseline PTA, DPOAEs and BERA those conducted after 12 months of period.

MEASUREMENT OF HbA1C-

- HbA1c is formed by nonenzymatic attachment of glucose to the N-terminal valine of HbA molecule of beta -chain. This covalent linkage changes the molecule’sstructure and charge;properties that is explored by following methods-
- Ion exchange chromatography.
- Immunoassay
- Electrophoresis
- High performance liquid chromatography
- It is measured as a percent of total haemoglobins.

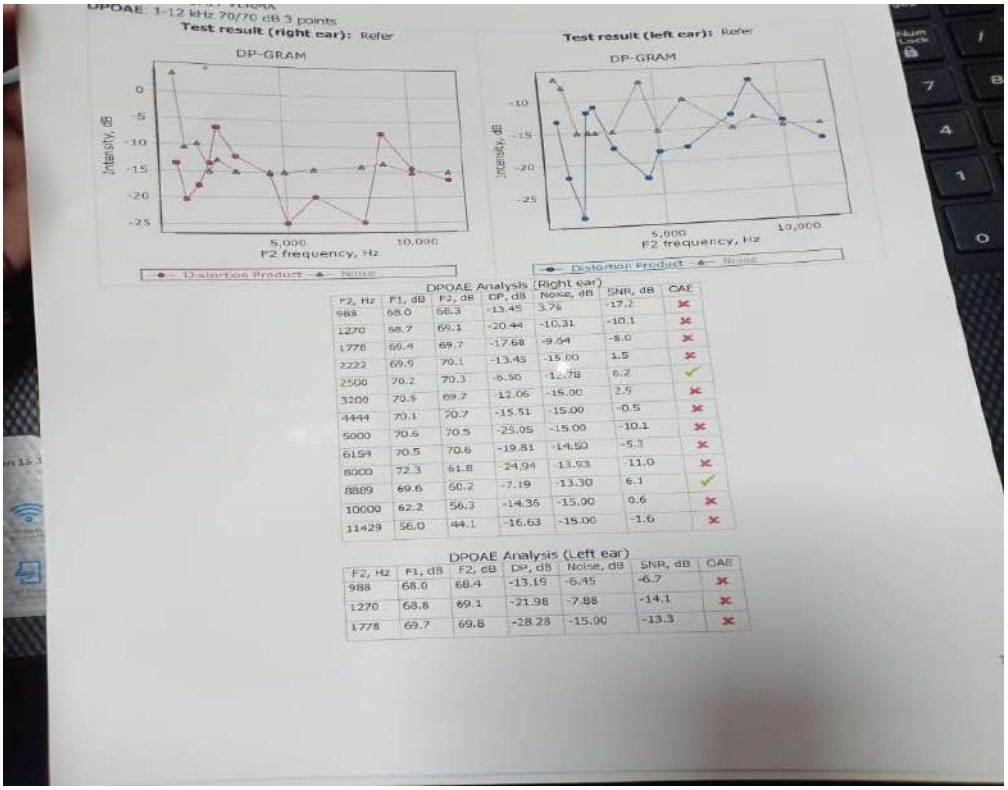
- Data was compiled and Master chart was made detailing all the relevant variables and appropriate statistical tests were applied for analysis.



NEUROAUDIO OTOACOUSTIC EMISSION AND BERA EQUIPMENT

OTOACOUSTIC EMISSION PROCEDURE

DIFFERENT SIZES OF EAR TIPS FOR OTOACOUSTIC EMISSION PROBE



DPOAE 1-12 kHz 70/70 dB 3 points

Test result (right ear): Refer

DP-GRAM

Test result (left ear): Refer

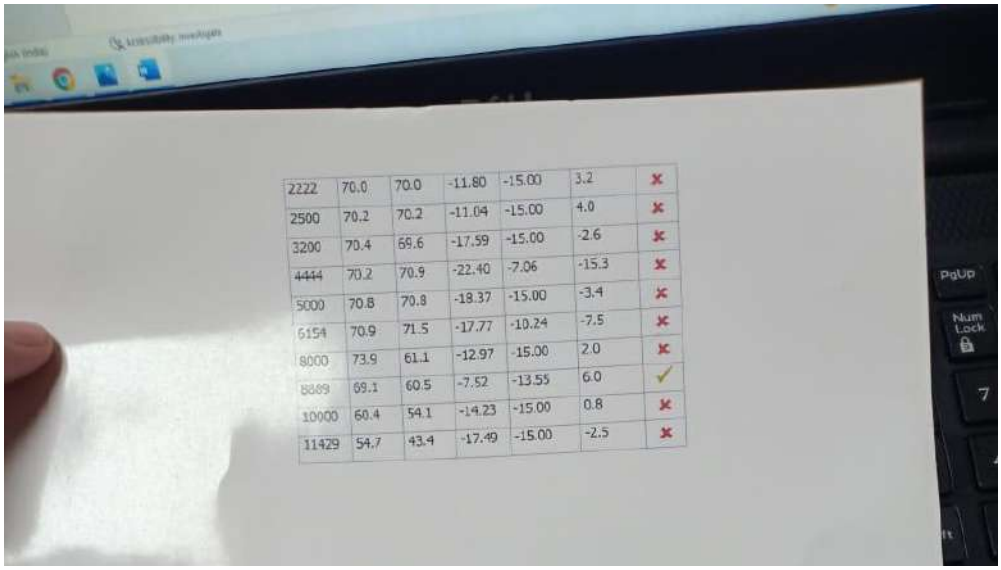
DP-GRAM

DPOAE Analysis (Right ear)

F2, Hz	F1, dB	F2, dB	DP, dB	Noise, dB	SNR, dB	CAE
988	68.0	68.3	-13.45	3.75	-17.2	✗
1270	68.7	69.1	-20.44	-10.31	-10.1	✗
1778	69.4	69.7	-17.68	-9.64	-8.0	✗
2222	69.9	70.1	-13.45	-15.00	1.5	✗
2500	70.2	70.3	-6.56	-12.78	6.2	✓
3200	70.5	69.7	-12.06	-15.00	2.9	✗
4444	70.1	70.7	-15.51	-15.00	-0.5	✗
5000	70.6	70.5	-25.05	-15.00	-10.1	✗
6154	70.5	70.6	-19.81	-14.50	-5.3	✗
8000	72.3	61.8	-24.94	-13.93	-11.0	✗
8889	69.6	60.2	-7.19	-13.30	6.1	✓
10000	62.2	56.3	-14.36	-15.00	0.6	✗
11429	56.0	44.1	-16.63	-15.00	-1.6	✗

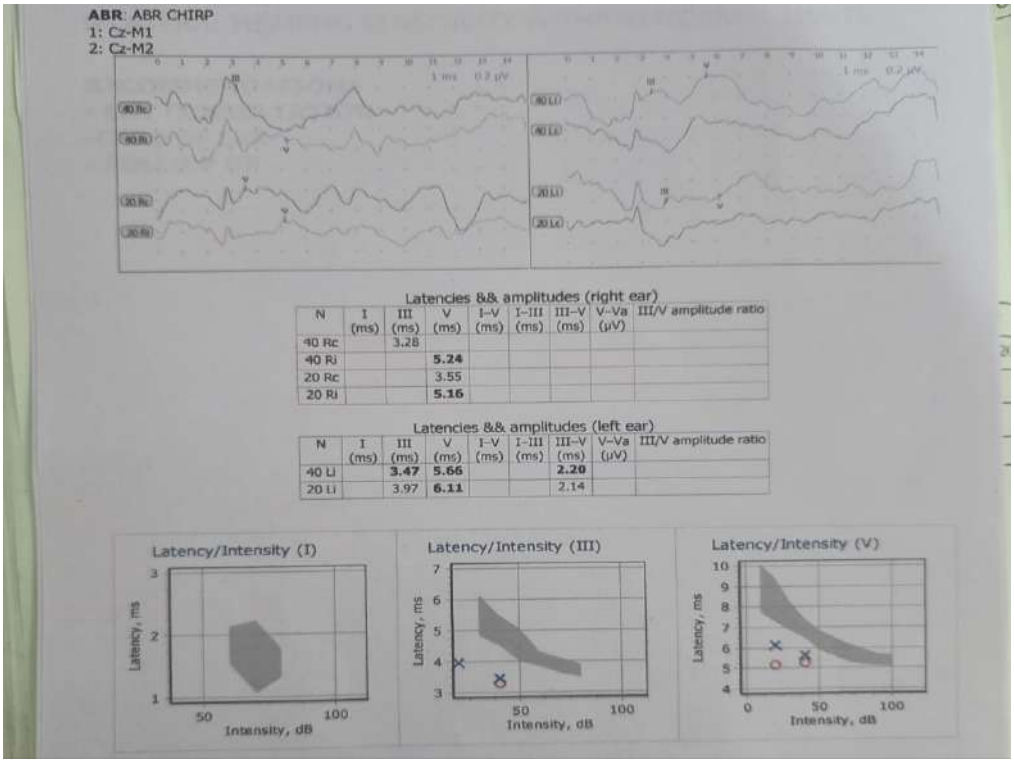
DPOAE Analysis (Left ear)

F2, Hz	F1, dB	F2, dB	DP, dB	Noise, dB	SNR, dB	CAE
988	68.0	68.4	-13.19	-6.45	-6.7	✗
1270	68.8	69.1	-21.98	-7.88	-14.1	✗
1778	69.7	69.8	-28.28	-15.00	-13.3	✗



2222	70.0	70.0	-11.80	-15.00	3.2	✗
2500	70.2	70.2	-11.04	-15.00	4.0	✗
3200	70.4	69.6	-17.59	-15.00	-2.6	✗
4444	70.2	70.9	-22.40	-7.06	-15.3	✗
5000	70.8	70.8	-18.37	-15.00	-3.4	✗
6154	70.9	71.5	-17.77	-10.24	-7.5	✗
8000	73.9	61.1	-12.97	-15.00	2.0	✗
8889	69.1	60.5	-7.52	-13.55	6.0	✓
10000	60.4	54.1	-14.23	-15.00	0.8	✗
11429	54.7	43.4	-17.49	-15.00	-2.5	✗

OTOACOUSTIC EMISSION GRAPH



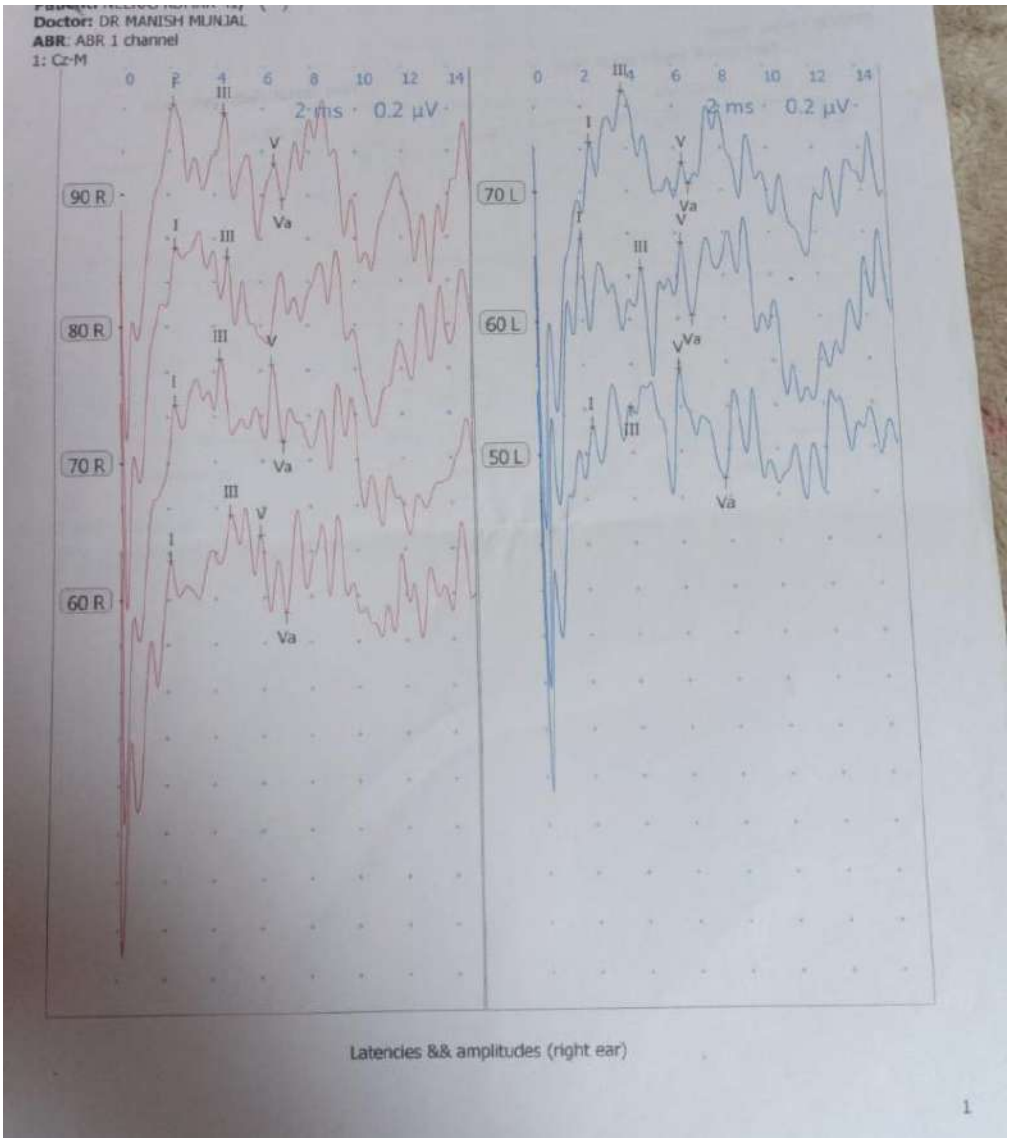
Latencies && amplitudes (right ear)

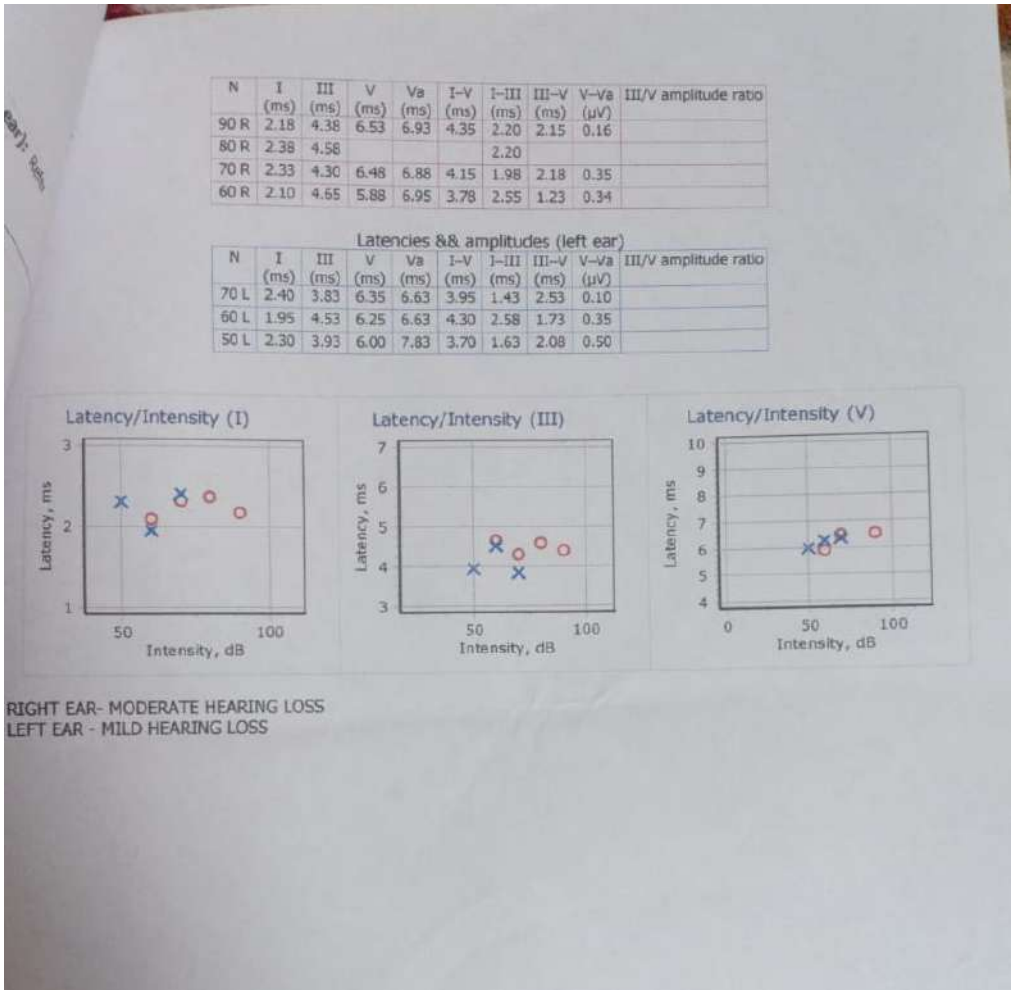
N	I (ms)	III (ms)	V (ms)	I–V (ms)	I–III (ms)	III–V (ms)	V–Va (µV)	III/V amplitude ratio
40 Rc		3.28						
40 Ri			**5.24**					
20 Rc			3.55					
20 Ri			**5.16**					

Latencies && amplitudes (left ear)

N	I (ms)	III (ms)	V (ms)	I–V (ms)	I–III (ms)	III–V (ms)	V–Va (µV)	III/V amplitude ratio
40 Li		**3.47**	**5.66**			**2.20**		
20 Li		3.97	**6.11**			2.14		

NORMAL BERA GRAPH


Doctor: DR MANISH MUNJAL
ABR: ABR 1 channel
1: Cz-M
2 ms 0.2 µV
90 R
80 R
70 R
60 R
70 L
60 L
50 L
I
III
V
Va
Latencies && amplitudes (right ear)
1



N	I (ms)	III (ms)	V (ms)	Va (ms)	I–V (ms)	I–III (ms)	III–V (ms)	V–Va (μV)	III/V amplitude ratio
90 R	2.18	4.38	6.53	6.93	4.35	2.20	2.15	0.16	
80 R	2.38	4.58				2.20			
70 R	2.33	4.30	6.48	6.88	4.15	1.98	2.18	0.35	
60 R	2.10	4.65	5.88	6.95	3.78	2.55	1.23	0.34	

Latencies && amplitudes (left ear)

N	I (ms)	III (ms)	V (ms)	Va (ms)	I–V (ms)	I–III (ms)	III–V (ms)	V–Va (μV)	III/V amplitude ratio
70 L	2.40	3.83	6.35	6.63	3.95	1.43	2.53	0.10	
60 L	1.95	4.53	6.25	6.63	4.30	2.58	1.73	0.35	
50 L	2.30	3.93	6.00	7.83	3.70	1.63	2.08	0.50	

RIGHT EAR- MODERATE HEARING LOSS
LEFT EAR - MILD HEARING LOSS

BERA GRAPH SHOWING HEARING LOSS

OBSERVATIONS AND RESULTS

30 patients who fulfilled the inclusion and exclusion criteria were included in the study.The observation of different parameters of the study have been compiled.

(A) DEMOGRAPHIC PROFILE

TABLE (I)

AGE DISTRIBUTION OF PATIENTS

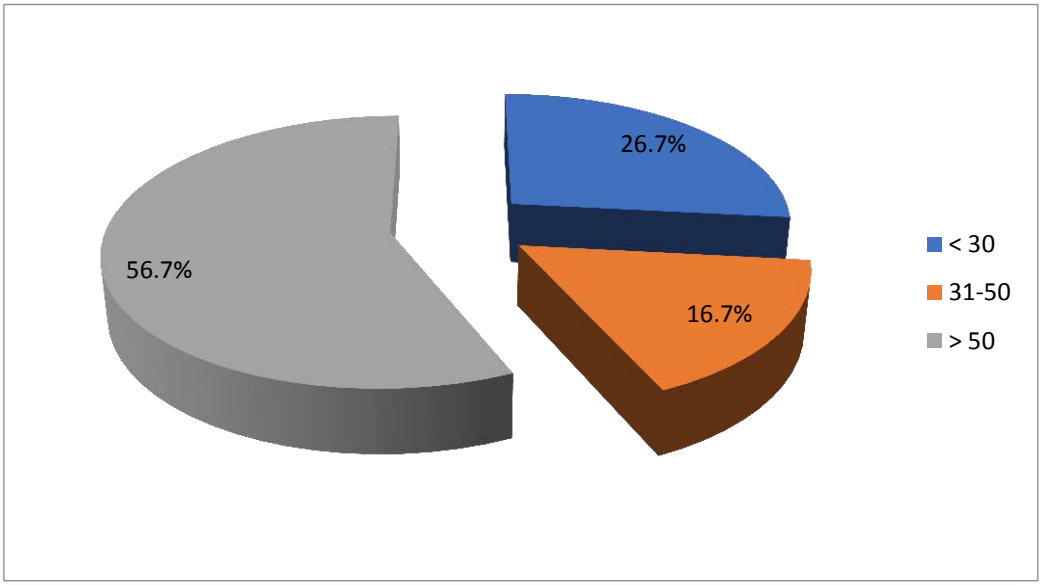
N=30

Age group	No. of cases	Percentage
< 30	8	26.7%
31-50	5	16.7%
> 50	17	56.7%
Total	30	100.0%

N =total number of patients, %=percentage of patients

FIGURE (I)

AGE DISTRIBUTION OF PATIENTS



Maximum number of patients were above the age of 50 years 17 (56.7%) followed by those below 30 years 8 (26.7%) .Only 5 (16.7%) were between 31-35 yrs.

TABLE (II)

GENDER DISTRIBUTION OF PATIENTS

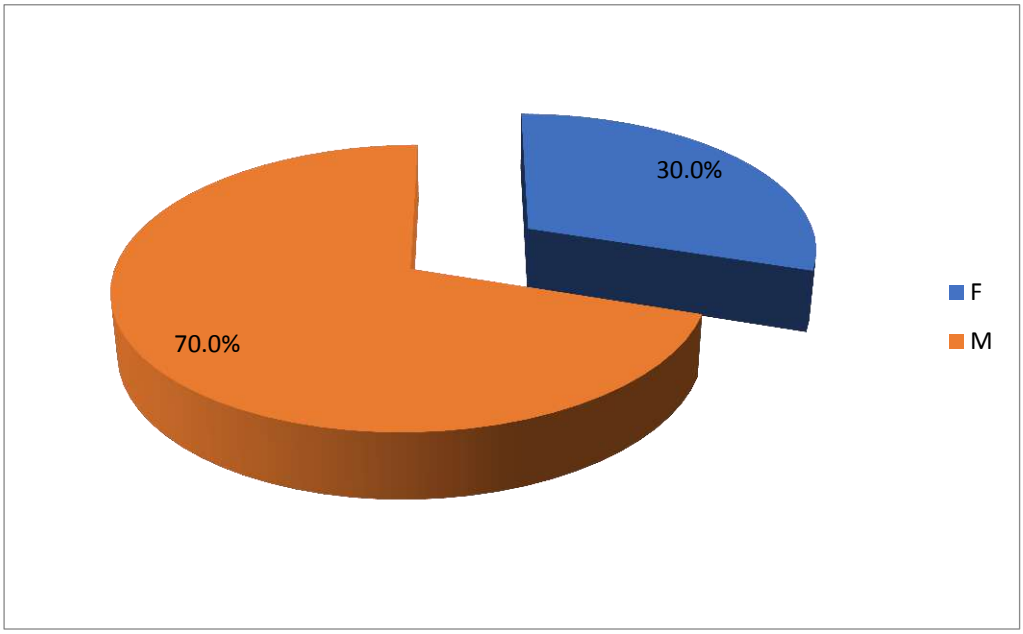
N=30

Sex	No. of cases	Percentage
F	9	30.0%
M	21	70.0%
Total	30	100.0%

N =total number of patients, %=percentage of patients

FIG (II)

GENDER DISTRIBUTION OF PATIENTS



Male patients (21) 70% outnumbered the females (9) 70%. The selection of patients was random and irrespective of gender.

Table (III)

TOPOGRAPHIC DISTRIBUTION OF THE PATIENTS

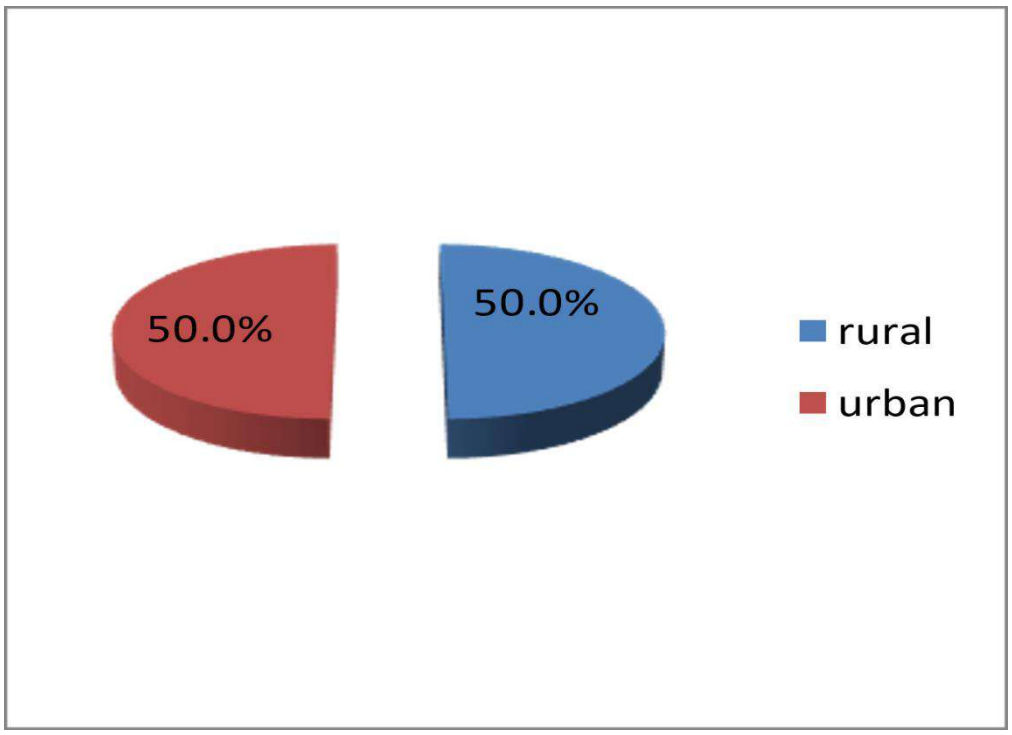
N=30

Locality	No. of cases	Percentage
Rural	15	50.0%
Urban	15	50.0%
Total	30	100.0%

N=30 total number of patients %=percentage of patients

FIG (III)

TOPOGRAPHIC DISTRIBUTION OF THE PATIENTS



15 (50%) patients were from rural and 15 (50%) from urban background.

(B) DIABETIC PROFILE

TABLE (IV)

DIABETIC STATUS OF PATIENTS

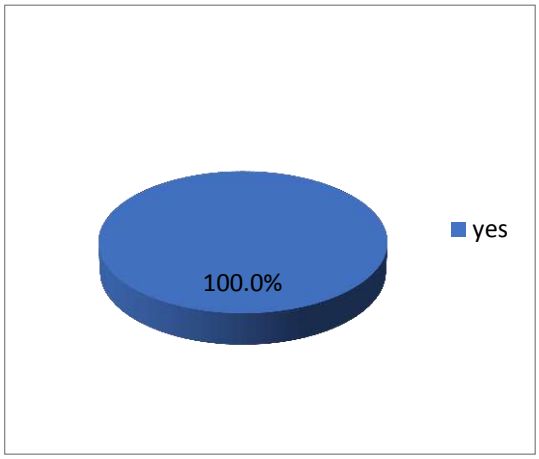
N=30

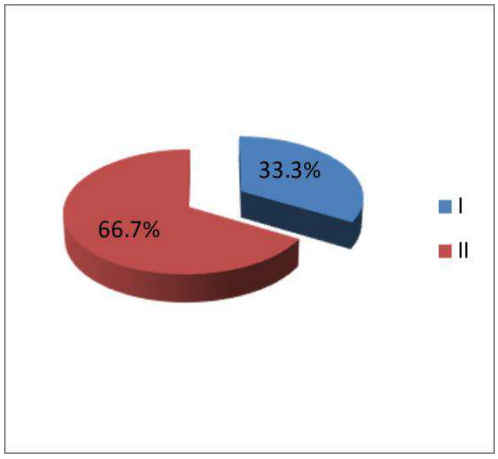
H/o Diabetes yes/no	No. of cases	Percentage
Yes	30	100.0%

N =total number of patients, %=percentage of patients

FIG (IV)

DIABETIC STATUS OF PATIENTS





N=30

Type of diabetes	No. of cases	Percentage
I	10	33.3%
II	20	66.7%
Total	30	100.0%

N =total number of patients, %=percentage of patients

30 (100%) patients were diabetic. Majority of patients were type II diabetic 20 (66.7%). Type I diabetic were only 10 (33.3%).

(C) AUDIOMETRIC EVALUATION

(i) Mean pure tone average

(ii) Otoacoustic emission

(iii) Brainstem evoked response audiometry

(i) MEAN PURE TONE AVERAGE ANALYSIS

30 (100%) patients underwent pure tone average analysis.

(TABLE V)

(A)CORRELATION OF PURE TONE AVERAGE THRESHOLD WITH HBA1C AND BLOOD GLUCOSE.

N =30

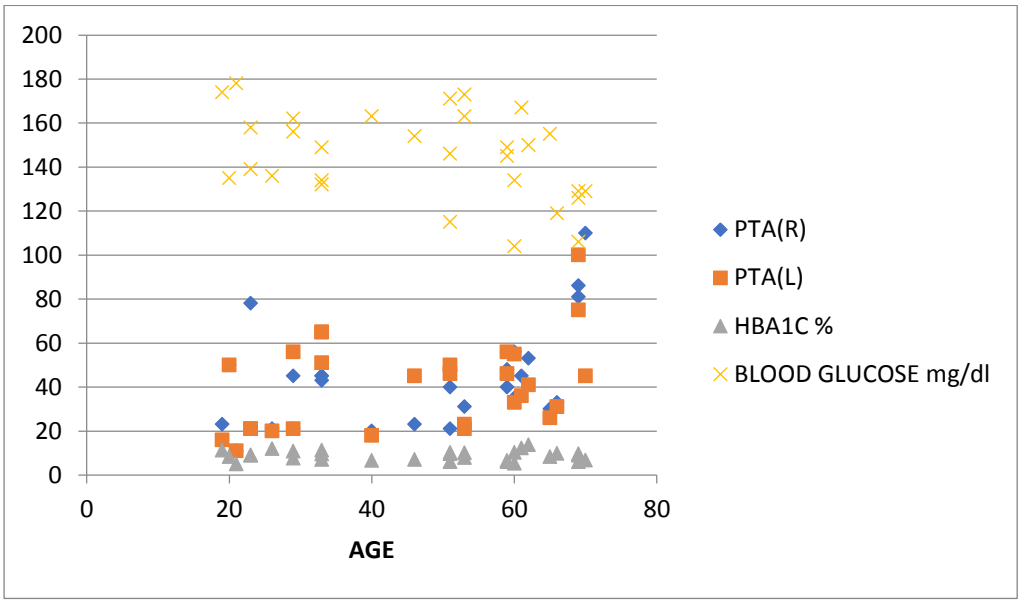
		Weight kg	**PTA (R)**	**PTA (L)**	**HBA1C %**	**BLOOD GLUCOSE mg/dl**
PTA(R)	Pearson Correlation	-0.136	1.000	0.604	-0.115	-0.577
	p-value	0.489		0.001	0.561	0.001
	N	28	28	28	28	28
PTA(L)	Pearson Correlation	-0.116	0.604	1.000	-0.153	-0.497
	p-value	0.557	0.001		0.438	0.007
	N	28	28	28	28	28

N =total number of patients, %=percentage of patients

The pure tone average threshold increases with increase in blood glucose,HBA1C.

FIG (V)

(A) CORRELATION OF PURE TONE AVERAGE THRESHOLD WITH HBA1C AND BLOOD GLUCOSEAND AGE



The pure tone average threshold increases with increase in blood glucose, HBA1C and age.

TABLE (VI)

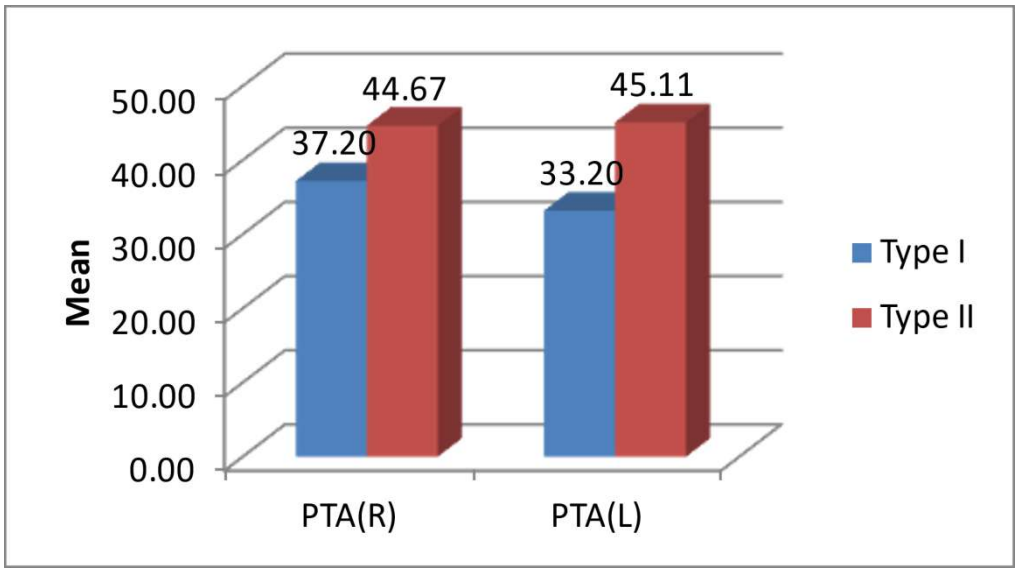
(B) CORRELATION OF PURE TONE AVERAGE WITH TYPE I AND TYPE II DIABETES

N=30

Type I		Type II		Z	p-value
Mean	SD	Mean	SD		
37.20	18.17	44.67	25.75	-0.809	0.426
33.20	16.23	45.11	22.14	-1.489	0.149

FIG (VI)

CORRELATION OF PURE TONE AVERAGE WITH TYPE I AND TYPE II DIABETES



In type II diabetes mean pure tone average was maximum (in right ear 44.67, left ear 45.11) as compared to type I diabetes (in right ear 37.20, p=0.426, left ear 33.20, p=0.149)

TABLE (VII)

(ii) OTOACOUSTIC EMISSION ANALYSIS

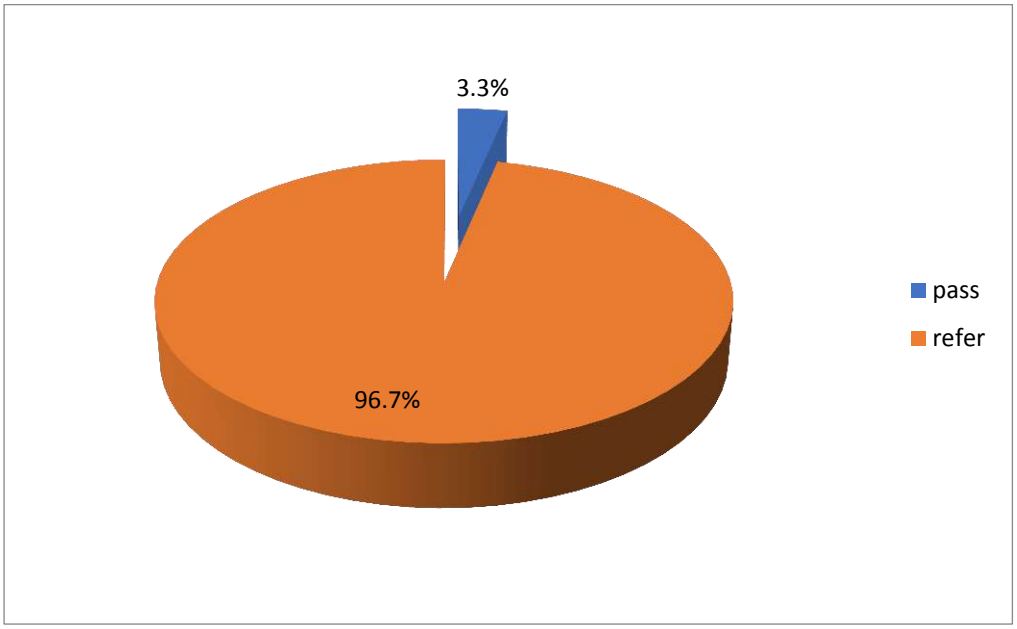
N=30

OAE	No. of cases	Percentage
Pass	1	3.3%
Refer	29	96.7%
Total	30	100.0%

N =total number of patients, %=percentage of patients

FIG (VII)

OTOACOUSTIC EMISSION ANALYSIS



In majority of patients, 29 (96.7%), otoacoustic emissions were absent. In only 3.3% patients otoacoustic emissions noted.

TABLE (VIII)

(A) CORRELATION OF OTOACOUSTIC EMISSIONS WITH AGE

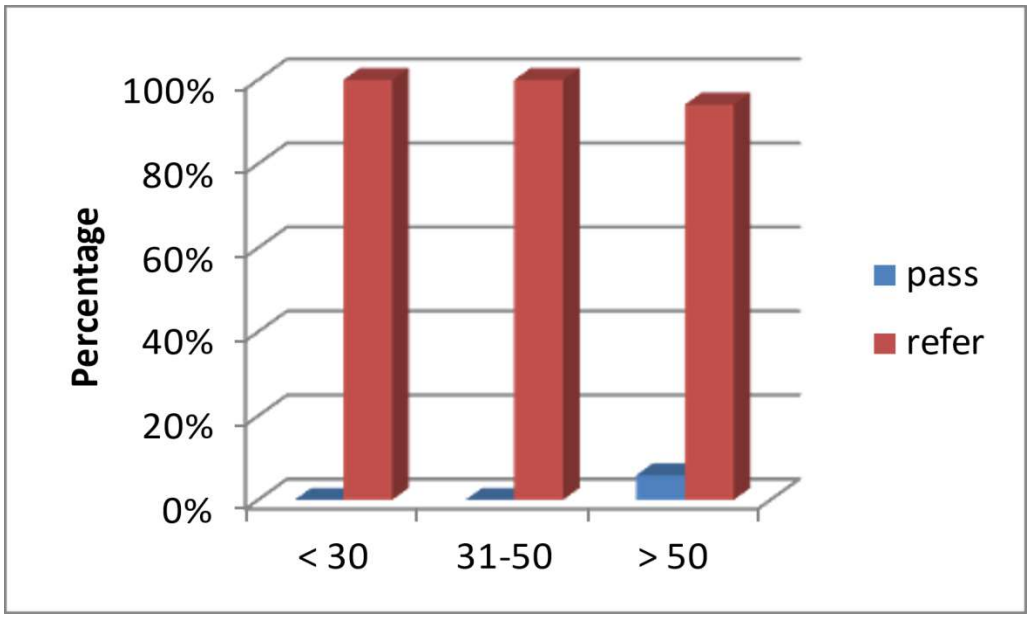
N=30

		OAE				Total
		Pass		Refer		
Age group	< 30	0	0%	8	100%	8
	31-50	0	0%	5	100%	5
	> 50	1	6%	17	94%	17
Total		1	3%	29	97%	30

N=total number of patients % = percentage of patients

FIG (VIII)

CORRELATION OF OTOACOUSTIC EMISSIONS WITH AGE



In majority of patients age group below 30 yrs and above 50 yrs otoacoustic emissions were absent and in 3% patients above 50 years age otoacoustic emissions were found.

TABLE (IX)

(B) **CORRELAION OF OTOACOUSTIC EMISSIONS WITH TYPE I AND TYPE II DIABETIC**

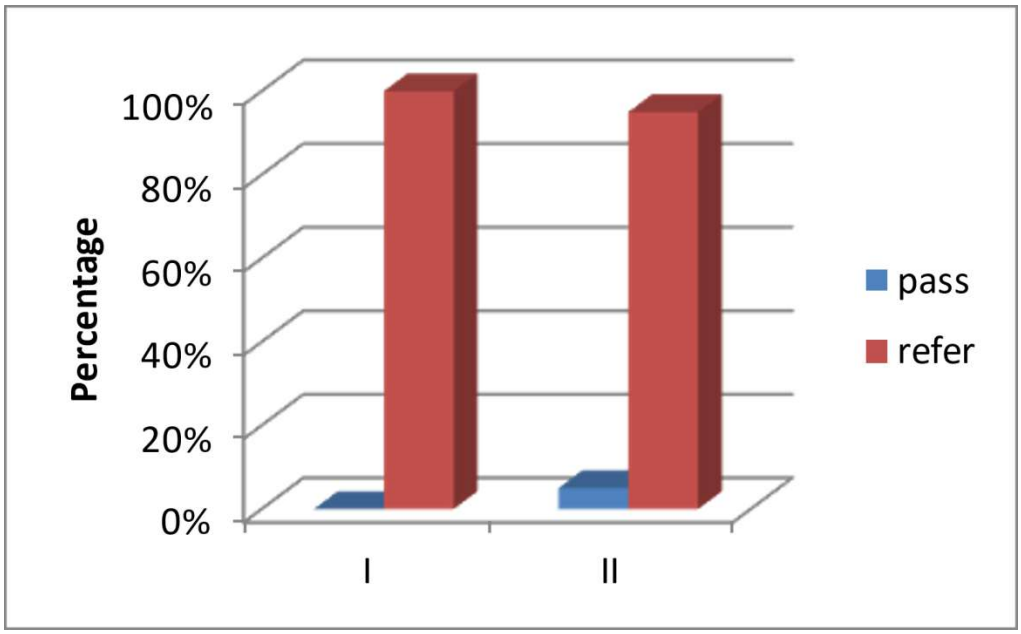
N=30

		OAE				Total
		Pass		Refer		
Type of diabetes	I	0	0%	10	100%	10
	II	1	5%	19	95%	20
Total		1	3%	29	97%	30

N=total number of patients % =percentage of patients

FIG (IX)

CORRELAION OF OTOACOUSTIC EMISSIONS WITH TYPE I AND TYPE II DIABETIC



In 10 (100%) patients of type I diabetes and 20 (95%) patients of type II diabetes otoacoustic emissions were absent.

TABLE (X)

(C) CORRELATION OF OTOACOUSTIC EMISSIONS WITH HBA1C

	Spearman rho	HBA1C
DPOAE	Correlation coefficient	0.591
	P value	0.001
	N	29

DPOAE amplitude decrease with increase in HBA1C. (p=0.001)

N=29

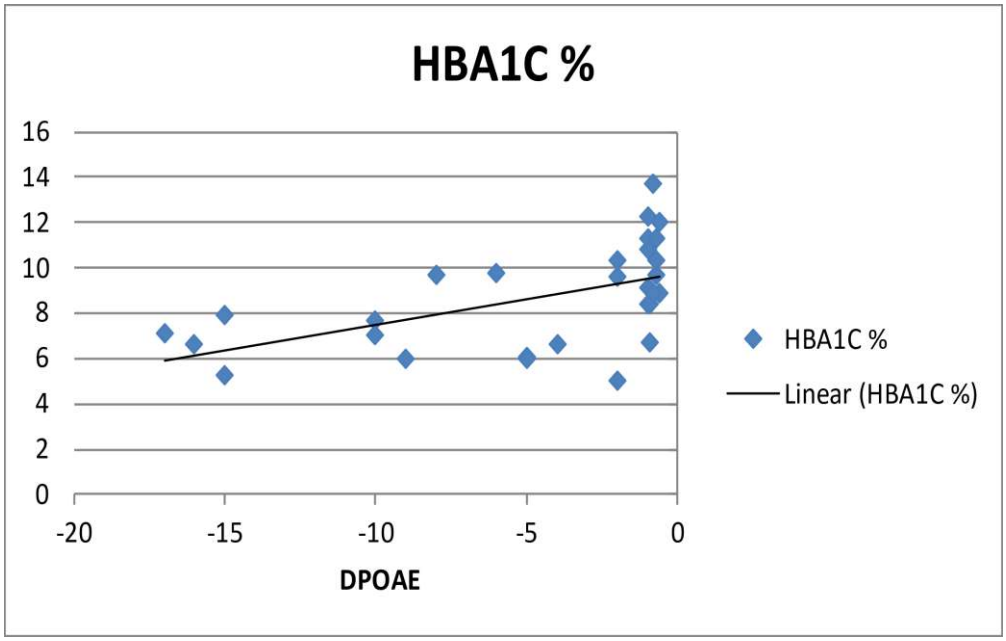
Frequency (kHz)	Mean DPOAE amplitude	Number of patients	Percentage
2 kHz	-9.1	5	17.2%
4kHz	-4.10	9	31.2%
6kHz	-1.9	12	41.3%
8kHz	-1.3	3	10.3%

N=total number of patients % =percentage of patients

Majority of patient 12(41.3%) had reduced amplitude of DPOAE at 6 kHz frequency followed by 9(31.2%) at 4kHz and 5(17.2%) at 2 kHz with minimal number 3 (10.3%) at 8kHz

FIG (X)

CORRELATION OF OTOACOUSTIC EMISSIONS WITH HBA1C



Scattered plot shows the relationship between amplitude of DPOAE and HbA1C (X axis shows the HbA1C levels and Y axis shows the mean DPOAE amplitude measure at 2 kHz, 4kHz, 6kHz and 8kHz).Mean DPOAE amplitude decrease with increase in HBA1C.

TABLE (XI)

(D) CORRELATION OF OTOACOUSTIC EMISSION WITH GENDER

N=29

Frequency (kHz)	Mean DPOAE amplitude	Male	females	Percentage
2 kHz	-9.1	3	2	17.2 %
4kHz	-4.10	7	2	31.2 %
6kHz	-1.9	9	3	41.3%
8kHz	-1.3	2	1	10.3%
		21(72.4%)	8 (27.5%)	100%

N=total number of patients % =percentage of patients.

Majority 21(72.4%) were males with reduced amplitude of DPOAE females 8 (27.5%). Maximum subjects (72.4%) were males with reduced amplitude of DPOAEs at a frequency of 6kHz.

TABLE (XII)

(iii) BERA ANALYSIS

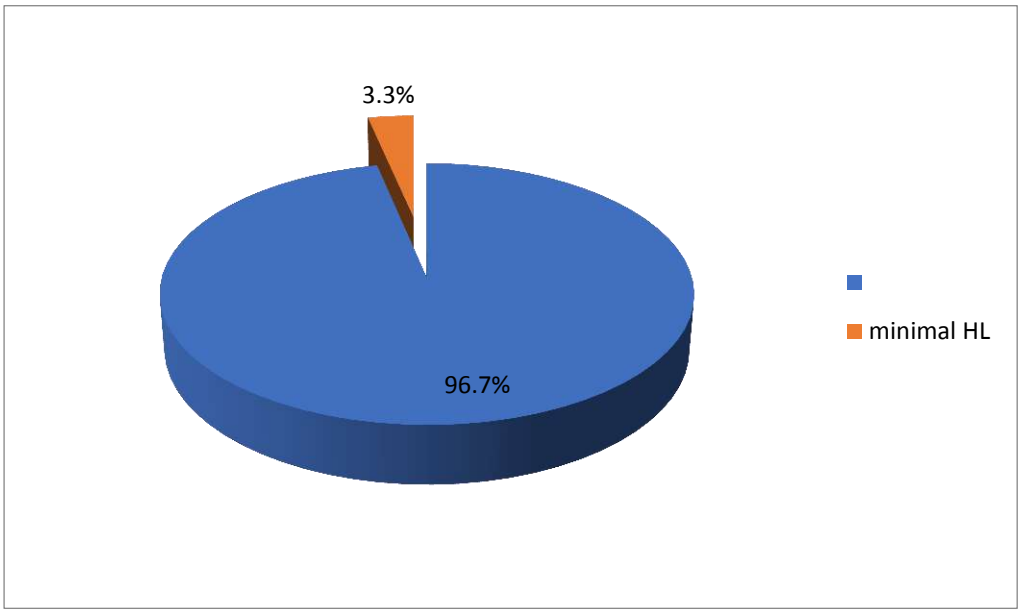
N=30

BERA	No. of cases	Percentage
	29	96.7%
Increased wave V Letency	1	3.3%
Total	30	100.0%

N =total number of patients, %=percentage of patients

FIG (XI)

BERA ANALYSIS



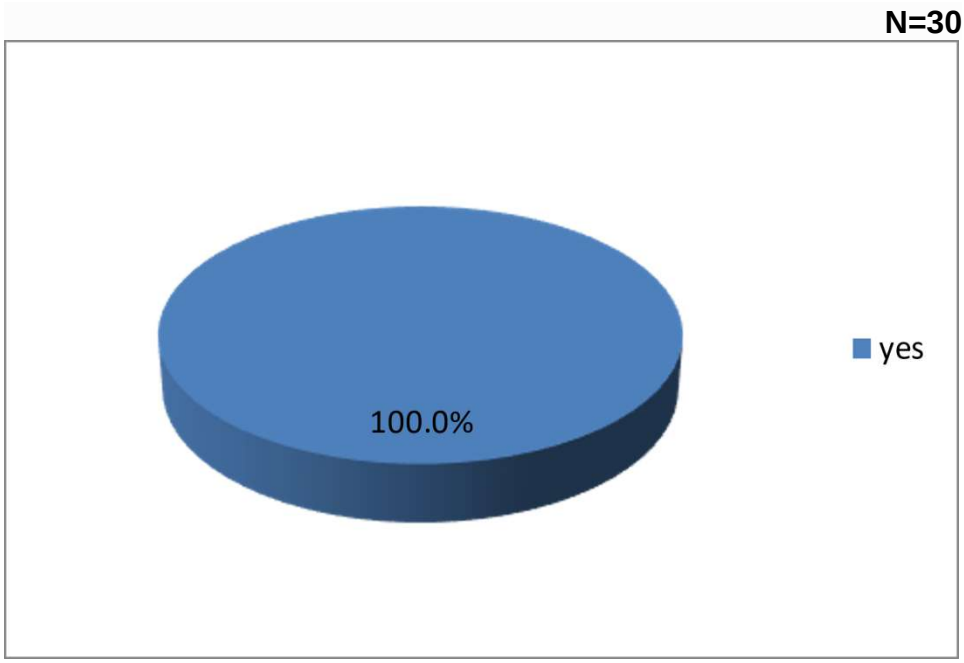
Out of 30 (100 %) patients of study group 1(3.3%) had retro cochlear involvement.

(D) CLINICAL PROFILE

FIG (XII)

(i) HISTORY OF IMPAIREMENT OF HEARING

Hearing loss with diabetes mellitus with type I and type II



N= total number of patients % =percentage of patients

Majority 30 (100%) patients were diabetic with impairement of hearing.

TABLE (XIII)

FAMILY HISTORY OF SENSORINEURAL HEARING LOSS

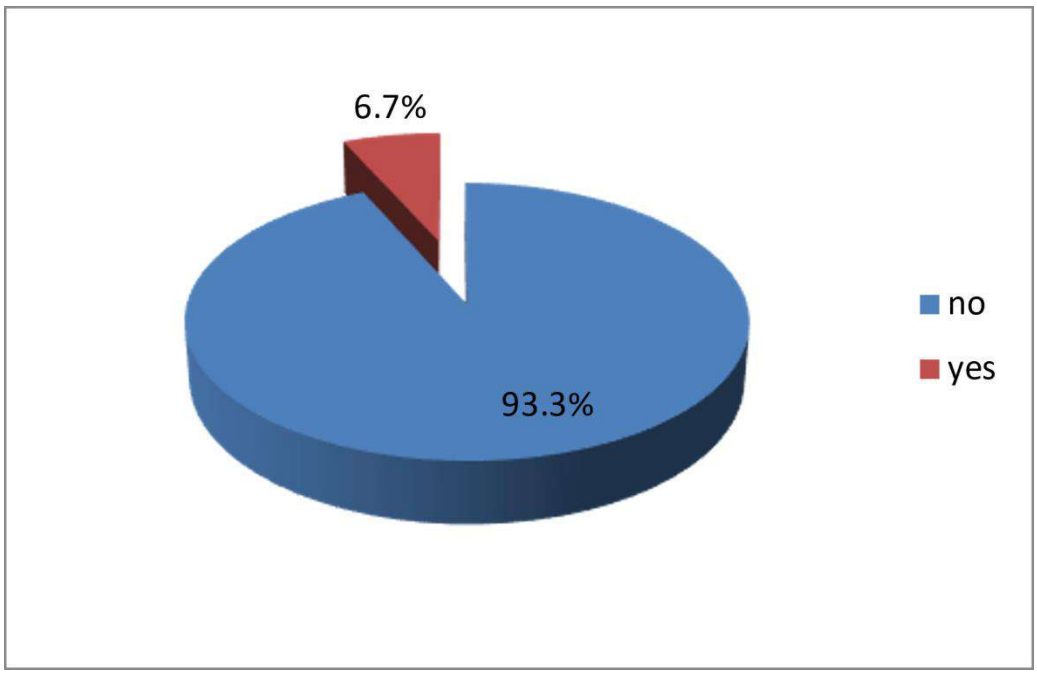
N=30

Family H/o Of SNHL	No. of cases	Percentage
No	28	93.3%
Yes	2	6.7%
Total	30	100.0%

N= total number of patients % =percentage of patients

FIG (XIII)

FAMILY HISTORY OF SENSORINEURAL HEARING LOSS



Majority of patients 30 (93.3%)had no family history of sensorineural hearing loss.

TABLE (XIV)

HISTORY OF EAR DISCHARGE & INTAKE OF OTOTOXIC DRUGS

N=30

H/O Ear d/c intake of ototoxic drugs	No. of cases	Percentage
No	21	70.0%
Yes	9	30.0%
Total	30	100.0%

N =total number of patients, %=percentage of patients

In majority, 21 (70%) patients had no history of ear discharge nor intake of ototoxic drugs.

TABLE (XV)

HISTORY OF TINNITUS

N=30

Tinnitus	No. of cases	Percentage
No	14	46.7%
Yes	16	53.3%
Total	30	100.0%

N =total number of patients, %=percentage of patients

Out of 30 majority of patients 16(53.3%) had history of tinnitus.

TABLE (XVI)

HISTORY OF HEAD INJURY

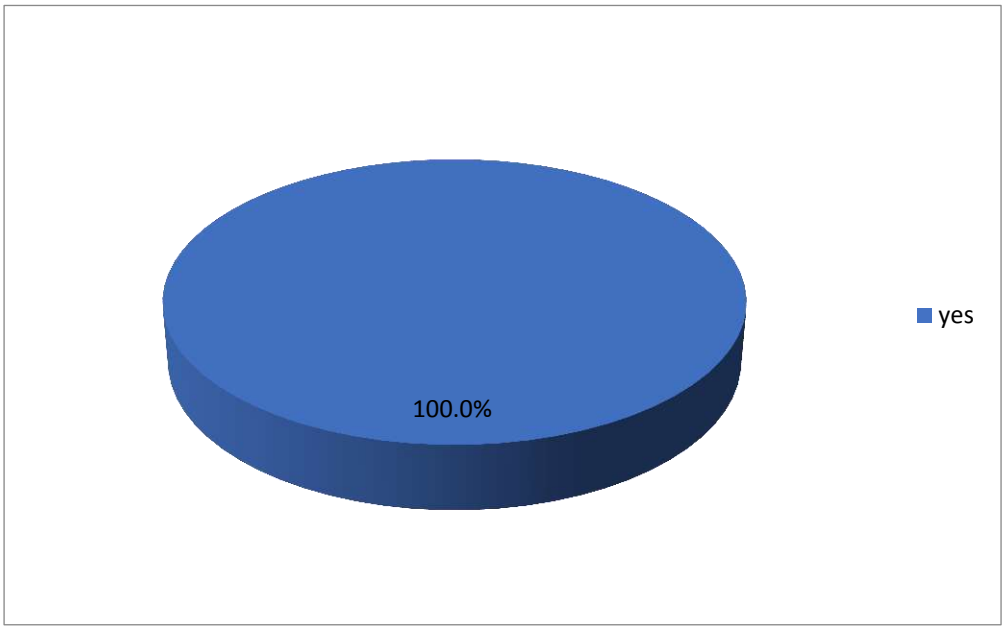
N=30

Head injury	No. of cases	Percentage
No	30	100.0%

N =total number of patients, %=percentage of patients

FIG (XIV)

HISTORY OF HEAD INJURY



Out of 30 (100%) patients none had history of head injury.

TABLE (XVII)

HISTORY OF EXPOSURE TO LOUD NOISE

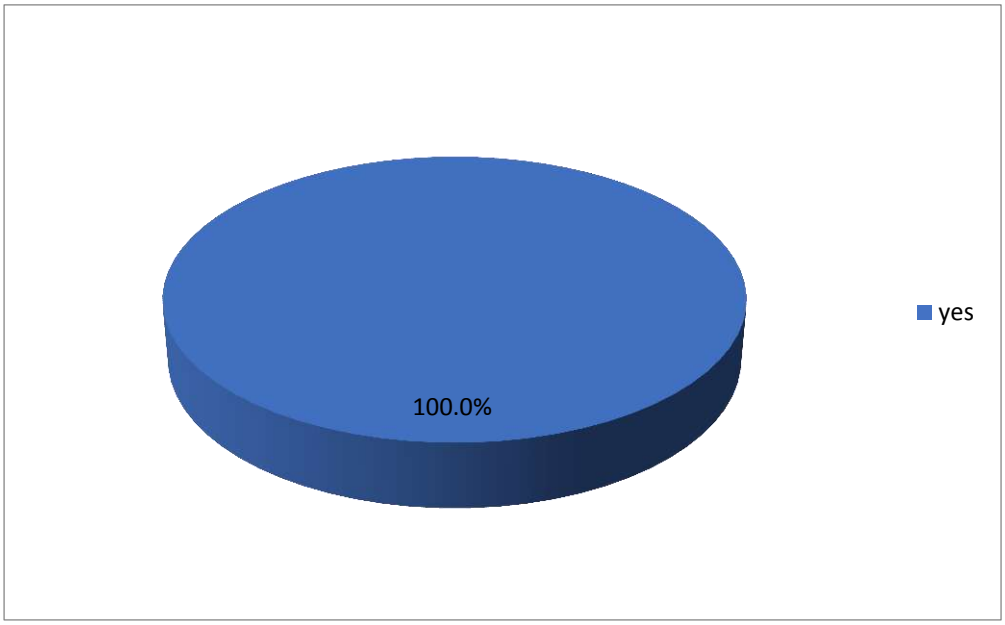
N=30

Exposure to loud noise	No. of cases	Percentage
No	30	100.0%

N =total number of patients, %=percentage of patients

FIG (XV)

HISTORY OF EXPOSURE TO LOUD NOISE



Amongst the analysed 30 (100%) patients none had history of exposure to loud noise.

TABLE (XVIII)

STATUS OF EXTERNAL AUDITORY *CANAL*

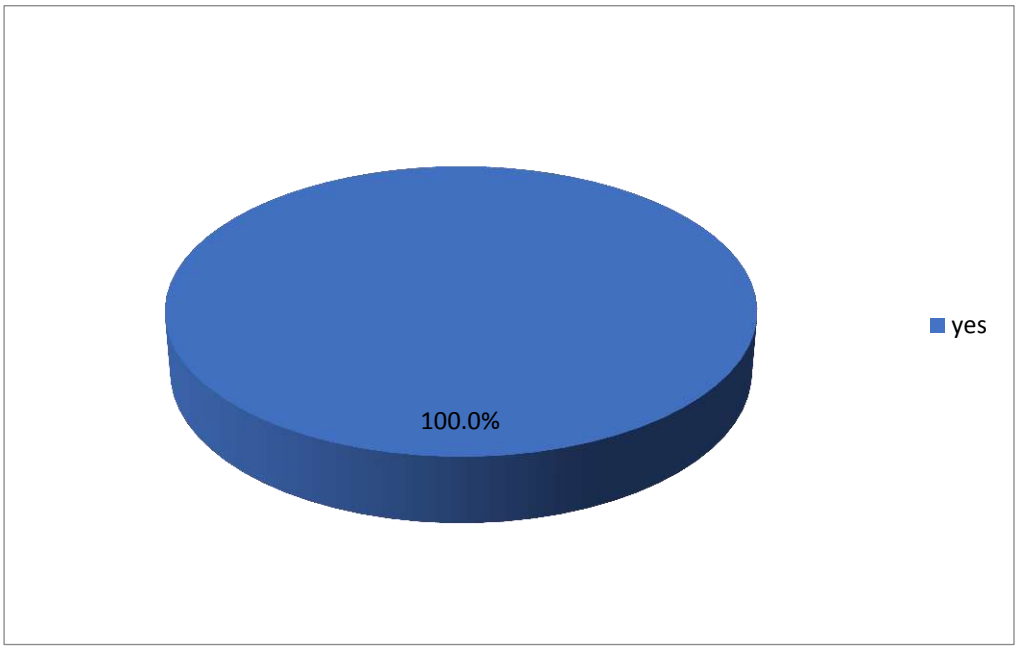
N=30

External appearance	No. of cases	Percentage
Normal	30	100.0%

N =total number of patients, %=percentage of patients

FIG (XVI)

STATUS OF EXTERNAL AUDITORY *CANAL*



In all 30 (100%) patients external auditory canal was normal.

TABLE (XIX)

STATUS OF TYMPANIC MEMBRANE

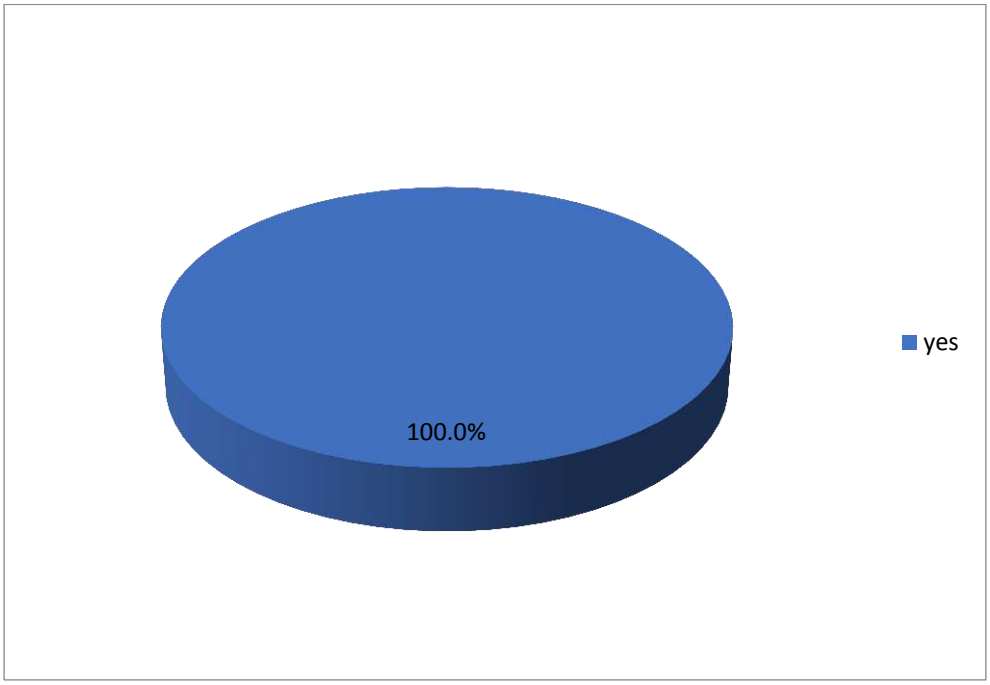
N=30

TM	No. of cases	Percentage
Intact	30	100.0%

N =total number of patients, %=percentage of patients

FIG (XVII)

STATUS OF TYMPANIC MEMBRANE



In all 30 patients tympanic membrane was intact.

TABLE (XX)

MOBILITY OF TYMPANIC MEMBRANE

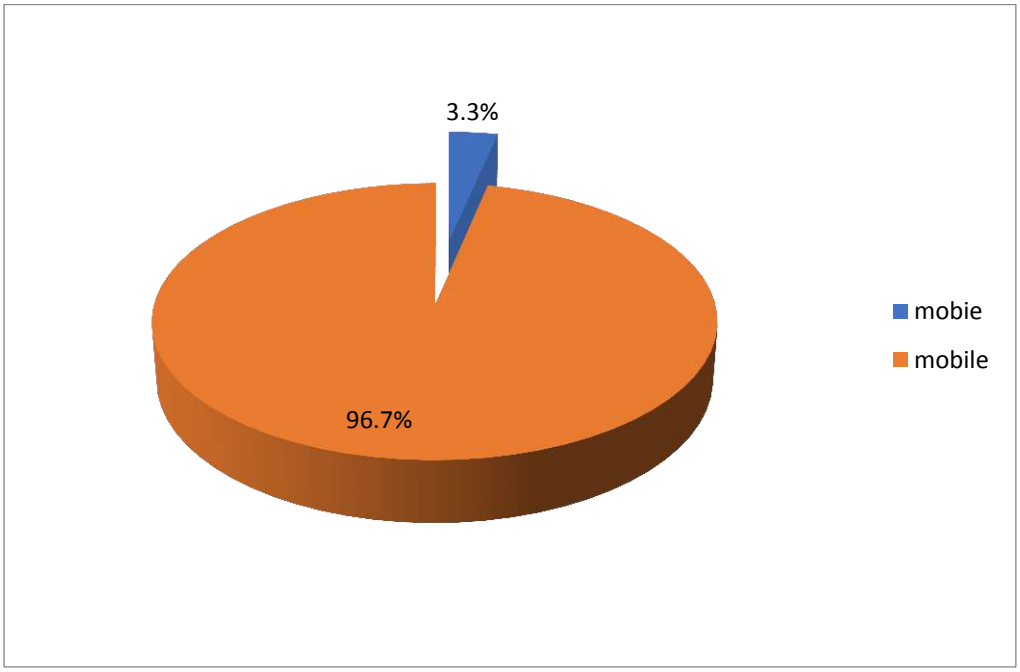
N=30

Mobility	No. of cases	Percentage
Immobile	1	3.3%
Mobile	29	96.7%
Total	30	100.0%

N =total number of patients, %=percentage of patients

FIG (XVIII)

MOBILITY OF TYMPANIC MEMBRANE



In majority of patients 29 (96.7%) tympanic membrane was mobile.

TABLE (XXI)

CONE OF LIGHT

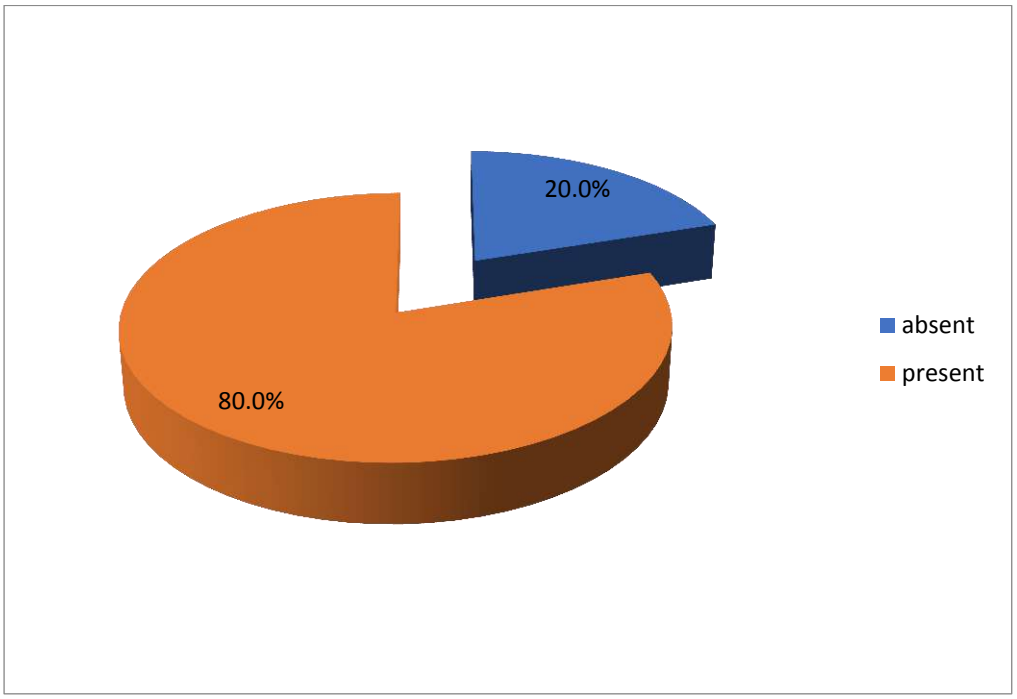
N=30

COL	No. of cases	Percentage
Absent	6	20.0%
Present	24	80.0%
Total	30	100.0%

N =total number of patients, %=percentage of patients

FIG (XIX)

CONE OF LIGHT



In majority of patients 24(80%) cone of light was present.

DISCUSSION

DEMOGRAPHIC PROFILE

AGE

In the present study, majority of patients were in age group above 50 years of age (56.7%) followed by 31 -50 years (16.7%) and below 30 years (26.7 %).

Youngest was 19 years of age and eldest was 70 years of age.

The Adriana Mia et al [11] 2005 53 patients study of diabetic patients with hearing loss, majority of patients were above 40 years.

Spankovich C et al [52] study had 40 young adults aged 18-28 years who were type-1 diabetic.

GENDER

In our 30 subject study, male patients 21 (70%) outnumbered the females 9(30%).

Mousavi SHG et al [87] study of 250 diabetic mellitus patients, had 158 (63.2%) females and 92 (36.8%) males.

Kautzky-Willer A et al [89] 1940 swedish study had a male predominance , with a male to female ratio up to 1.4 in the age group 10–55 years.

In the Ottaviani F et al study [56] of 60 diabetic patients, male patients 35 outnumbered the female patients 25.

CLINICAL PROFILE

HEARING IMPAIREMENT

In the present study 30 adult patients were diabetic with complaint of hearing loss (100%).6.7% had family history of sensorineural hearing loss. Majority 28(93.3%) had no family history of sensorineural hearing loss.

In the Al-Rubeaan K et al [72] 157 30-60 yrs diabetic subjects study, aged 30-60 years there were, 77 with hearing loss in both ears

(49.0%), 13 had involvement of right ear only (8.3%) , 14 of left ear only (8.9%); and 53 (33.8%) had normal hearing. In the 181 ears with sensorineural hearing loss, 90 had mild loss (49.7%), 69 moderate loss (38.2%), 16 severe loss (8.8%) and 6 had profound loss (3.3%).

Akinpelu OV et al [48] study concluded that the incidence of hearing loss ranged between 44% and 69.7% for type 2 diabetics; significantly higher than in controls. The mean PTA (pure tone audiometry) thresholds were greater in diabetics than in controls for all frequencies.

Hou Y, Xiao X et al [50] analysis of diabetic patients demonstrated a hearing deficit with elevated hearing thresholds in right ear as well as left ear when compared to healthy controls.

ASSOCIATED SYMPTOMS

TINNITUS

In the present study 53.3% patient had history of tinnitus. Approximately 100 % patients had negative history of head injury and exposure to loud noise.

Somogyi A, Rosta K et al [71] study reported 34% diabetic patients with tinnitus.

Mousavi SHG et al [87] study concluded that 66 % of the 250 diabetic patientshad tinnitus.

Ameen SA et al [88] study concluded prevalence of tinnitus among diabetic patients to be about 24.1% (32/133).

EAR DISCHARGE AND INTAKE OF OTOTOXIC DRUGS

In our study majority 21 (70%) patients had no history of ear discharge and intake of ototoxic drugs. Moreover none of the 30(100%) patients had history of head injury, exposure to loud noise.

STATUS OF EXTERNAL AUDITORY CANAL AND TYMPANIC MEMBRANE

In our study of 30 (100%) patients the external auditory canal and tympanic membrane was normal, in 29(96.7%) the tympanic membrane was mobile. Cone of light was present in 24 (80%) patients.

PURE TONE AUDIOMETRIC ASSSSMENT

In our study diabetic patients revealed higher hearing threshold at high frequencies. The mean pure tone average of patients with type I diabetes was in right ear 37.20,left ear 33.20. In type II diabetes was 44.67 in right ear, 45.11 in left ear.As the HBA1C increases mean pure tone average threshold increases(p=0.001)

Lasagni A, Giordano P et al study[73] in diabetes patients showed significantly higher mean hearing thresholds.

Botelho CT, Carvalho et al [53] study of 40 diabetic patients showed sensorineural hearing loss, affecting frequencies of 6000 and 8000 Hz, which was found only in type 1 DM subjects when compared to the controls .

OTOACOUSTIC EMISSIONS

In our 30 subjects study only 3.3 % of patients had otoacoustic emissions. With increase in HBA1C (p= 0.001)DPOAE amplitude decreases. Majority of patient 12(41.3%) had reduced amplitude of DPOAE at 6 kHz frequency followed by 9(31.2%) at 4kHz and 5(17.2%) at 2 kHz with minimal number 3 (10.3%) at 8kHz Majority 21(72.4%) were males with reduced amplitude of DPOAE with females 9(27.5%).Maximum subjects 21(72.4%) were males with reduced amplitude of DPOAE at a frequency of 6kHz.

Otoacoustic emissions reflect the function of cochlear outer hair cells, which are responsible for qualitative sound perception. Hence, damage to the outer hair cells results in subclinical hearing dysfunction, as detected in our study.

Ottaviani F, Dozio N et al [74] conducted the study Seventeen patients (28.3%) had no OAEs in at least one ear and 10% in both ears. Participants were 40adolescents with DM1 and 40 healthy subjects. DPOAE amplitude at medium frequencies (2.8–4 kHz) were significantly lower in patients with diabetes.

Dąbrowski M et al [75] study reported the amplitude of otoacoustic emissions was significantly lower in the diabetic patients.

Botelho CT et al [53] detected cochlear damage was detected by DPOAE responses with 32% belonging to the diabetic group, vs. 3.7% in the control group.

BERA

In our study 3.3.% patients had prolonged wave V latency and a retrocochlear involvement .As there was only one diabetic patient there was no correlation with increase in HBA1C value.

Ottaviani F, Dozio N et al [74] study ABR, in addition to delay in waves I, III and V, observed the appearance of a visible wave IV in patients with diabetes compared with controls.

Dąbrowski M et al [75] study among veterans with type 2 diabetes reported elevated hearing threshold and prolonged latencies of wave V and interval I-V especially in younger patients (below 50 years) who had been treated with insulin, and having other diabetic complications

In Akinpelu OV et al [48] study on Auditory brainstem responses (ABR) Wave V latencies were statistically significantly longer in diabetics.

DIABETIC PROFILE

In our study group, 30 (100%)patients were diabetic. Majority of patients were type II diabetic 20 (66.7%) with type I diabetic percentage of 33.3%.

Botelho CT et al study [49]included 40 adolescents with DM1 and 40 healthy subjects. Eren E et al [82] study involved 40 type 2 diabetic patients and 24 healthy volunteer.Samocha-Bonet D et al [90] study concluded that both type 1 and type 2 diabetes have been associated with hearing loss.

COMPARATION WITH VARIOUS STUDIES

Parameter	Study group	Study period	Number of subjects	Outcome
DEMOGRAPHIC PROFILE				
Age	Dmch study	2021-2022	30	Minimum age 19 years and maximum age 70 years.
	K.E. Bainbridge et al[76]	1999 to 2004	514	Mean years of age were between 20- 69 years
Gender	Dmch study	2021-2022	30	21:9(male<females)
	Weiwei Wang, Chen Zhang et al [77]	2011-2018	16410	Hazard ratio for hearing loss in women was 1.20 and in men 0.97
Locality	Dmch study	2021-2022	30	50% patients were from urban area.
	Pradeepa R et al[86]	2021	-	The prevalence of diabetes was higher in urban areas (11.2%) compared to rural areas (5.2%).
Audiometric analysis (PTA)	Dmch study	2021-2022	30	30 diabetic patients had hearing loss with Mean PTA of patients with type I diabetes in right ear is 37.20 , left ear 33.20.Mean PTA of patients with type II diabetes in right ear is 44.67, left ear 45.11.
	D.F. Austin et al [78]	2009	165	165 diabetic patients had sensorineural hearing loss
	Wackym, Phillip et al[79]	1986	08	08 diabetic patients had sensorineural hearing loss.
	Zhi-Pan Teng MD et al [80]	2016	252	252 diabetic patient had sensorineural hearing loss
OAE	Dmch study	2021-2022	30	29 (97%) patient had no otoacoustic emissions

	Eren E et al [82]	2014	40	40 diabetic patients had decreased OAE amplitude.
	Madhavan Balakrishnan et al [83]	2021	90	In 60 diabetic patients (30 type 1 ,30 type 2 diabetic DPOAE amplitude reduction in both experimental groups (Type 1, Type 2 DM) is comparatively equal
	Ali Hilali et al[84]	2009	21	Mean TEOAE amplitude was found to be significantly reduced ($p < 0.001$) in the diabetic patients (mean 9.0 ± 1.7 dB SPL)
BERA	Dmch study	2021-2022	30	3.3 % patient had prolonged wave V latency.
	Olubunmi V. Akinpelu MD et al [81]	2014	24	Type 2 diabetic patients had prolonged ABR wave v latencies.
Diabetic profile	Dmch study	2021-2022	30	Diabetic patient with hearing loss had high levels of blood glucose and HbA1c
	Shen Y, Zheng Z et al [85]	2021	116	Higher HbA1c level associated with high risk of hearing loss in diabetic patients.

SUMMARY & CONCLUSION

CLINICAL STUDY

- Diabetes is a chronic health condition that causes considerable morbidity and mortality.
- Both type 1 and type 2 diabetes have been associated with hearing loss secondary to changes in the organ of Corti and stria vascularis.
- Hearing Impairement include the combined contributions of hyperglycemia and oxidative stress to cochlear microangiopathy and auditory neuropathy.
- Quality of life improves in diabetic patients with control of glycemic index.
- Pure tone average shows high hearing threshold in higher frequencies with increase in HbA1c levels (p =0.001).
- With increase in HbA1C amplitude of recorded waves for OAE in 29 diabetics were significantly lower (p = 0.001) which attributed to disorder in the hair cells of the cochlea.
- Majority of patient 12(41.3%) had reduced amplitude of DPOAE at 6 kHz frequency followed by 9(31.2%) at 4kHz and 5 (17.2%) at 2 kHz with minimal number 3 (10.3%) at 8kHz Majority 21(72.4%) male had reduced amplitude of DPOAE with female number 9(27.5%).
- On BERA test increased latencies and significant wave V delay suggesting acoustic neuropathy .As there was only one diabetic patient there was no correlation with increase in HbA1c

CONCLUSION-

- In type 2 diabetic 6kHz was the frequency which was involved in maximal subjects.
- Male diabetic had higher incidence of involvement.
- PTA,OAE and BERA is a non invasive screening tool to assess the function of cochlea and vestibulocochlear nerve for early detection and timely management of hearing loss in diabetic patient.

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PROFORMA

Sr No.

Name of the patient..... Age..... Sex.....

MRD. No.....Weight.....

Address.....

.....

.....

HISTORY

1) H/O Diabetes

2) H/O decreased hearing

3) H/O ear discharge /earache/tinnitus....

4) H/O intake of any ototoxic drug.....

5) H/O head injury with documented temporal bone fracture....

6) H/O exposure to abnormally loud sounds....

7) Family history of SNHL.....

8) Any other mentionable findings....

Signature of Supervisor

Signature of Candidate

EXAMINATION OF THE EAR

External appearance.....

Otoscopic examination....

TM.....

Mobility....

COL.....

BASE LINE INVESTIGATION

1) PTA
2) OAE
3) BERA
4) BLOOD GLUCOSE
5) HBA1C

SR NO.	Otoacoustic Emissions	Pass	Refer

Signature of Supervisor

Signature of Candidate

Sr. No	MRD	Name	Age (Yrs)	Sex	Locality	Diabetes yes/no	Type I diabetes	type ii diabetes	Family H/o Of SNHL	H/O IOH	Ear Discharge	Intake of ototoxic drugs	Tinnitus	Head injury	Exposure to loud noise
1	1375145	SK	62	F	urban	yes	-	+	yes	yes	no	no	yes	no	no
2	1325625	AK	33	F	urban	yes	-	+	no	yes	yes	no	no	no	no
3	1348236	SC	20	M	rural	yes	+	-	no	yes	no	no	yes	no	no
4	1381321	GK	69	F	urban	yes	-	+	no	yes	no	no	yes	no	no
5	1380791	KK	51	M	urban	yes	-	+	no	yes	yes	no	no	no	no
6	944997	DK	66	F	rural	yes	-	+	yes	yes	no	no	no	no	no
7	1415677	BS	33	M	rural	yes	-	+	no	yes	no	no	yes	no	no
8	200597	KK	70	F	urban	yes	-	+	no	yes	yes	no	yes	no	no
9	69543	DS	60	M	urban	yes	-	+	no	yes	yes	no	no	no	no
10	1352744	PS	61	M	urban	yes	-	+	no	yes	no	no	yes	no	no
11	1243553	SK	53	F	rural	yes	-	+	no	yes	no	no	no	no	no
12	681860	SK	26	M	rural	yes	+	-	no	yes	no	no	no	no	no
13	1249583	SR	59	M	rural	yes	-	+	no	yes	yes	no	no	no	no
14	1378397	BS	66	M	urban	yes	-	+	no	yes	no	no	yes	no	no
15	1326134	SL	60	M	rural	yes	-	+	no	yes	no	no	no	no	no
16	1288357	SS	59	M	urban	yes	-	-	no	yes	yes	no	yes	no	no
17	1415677	BS	29	M	rural	yes	+	-	no	yes	yes	no	no	no	no
18	1318533	VD	69	F	urban	yes	-	+	no	yes	no	no	no	no	no
19	635597	BS	29	M	rural	yes	+	-	no	yes	no	no	yes	no	no
20	682654	GS	51	M	urban	yes	-	+	no	yes	no	no	yes	no	no
21	1008789	A	19	F	urban	yes	+	-	no	yes	no	no	yes	no	no
22	315777	MS	23	M	urban	yes	+	-	no	yes	no	no	yes	no	no
23	1181695	MG	53	M	rural	yes	-	+	no	yes	yes	no	no	no	no
24	1427529	GS	33	M	rural	yes	+	-	no	yes	no	no	no	no	no
25	1408681	JS	65	M	rural	yes	+	-	no	yes	yes	no	yes	no	no
26	1415677	BS	23	M	urban	yes	+	-	no	yes	no	no	yes	no	no
27	1380986	N	51	F	rural	yes	+	-	no	yes	no	no	yes	no	no
28	431301	RC	40	M	urban	yes	-	+	no	yes	no	no	yes	no	no
29	49133	MS	21	M	rural	yes	-	+	no	yes	no	no	no	no	no
30	654439	VK	46	M	rural	yes	-	+	no	yes	no	no	no	no	no

Sr. No	MRD	External apperance	TM	Mobility	COL	PTA(R/L)	OAE	DPOAE 2kHz	DPOAE4K4k HZ	DPOAE 6kHz	DPOAE 8KHz	BERA	HBA1C %	BLOOD GLUCOSE mg/dl
1	1375145	Normal	intact	mobie	present	53, 41	refer	-2.63	-1.83	-0.8	-3.9	-	13.7	150
2	1325625	Normal	intact	mobile	absent	45, 65	refer	-9.8	-7.4	-2.2	-13.7	-	9.6	149
3	1348236	Normal	intact	mobile	absent	50,50	refer	-2.93	-2.3	-1.9	-11.5	-	8.4	135
4	1381321	Normal	intact	mobile	absent	60 69	refer	-7.12	-7.5	-0.7	-12.8	-	9.7	126
5	1380791	Normal	intact	mobile	present	50 ,52	refer	-5.25	-3.5	-2.6	15.7	-	10.3	115
6	944997	Normal	intact	mobile	absent	81,75	refer	-1.83	-1.5	-0.6	-9.9	-	8.9	106
7	1415677	Normal	intact	mobile	absent	45,65	refer	-13.73	-11.4	-1.7	-16.7	-	11.3	134
8	200597	Normal	intact	mobile	absent	110,45	refer	-12.88	-10.9	-0.9	-15.7	-	6.7	129
9	69543	Normal	intact	mobile	present	35,33	refer	-5.25	-4.3	-0.7	-17.6	-	10.3	104
10	1352744	Normal	intact	mobile	present	45,36	refer	-6.5	-5.7	-1.3	-20.6	-	12.3	167
11	1243553	Normal	intact	mobile	present	31,23	pass	7.12	6.2	9.4	8.5	prolonged wave V latency	10.1	173
12	681860	Normal	intact	mobile	present	21,20	refer	-4.1	-3.12	-0.6	-22.3	-	12	136
13	1249583	Normal	intact	mobile	present	48,46	refer	-9.96	-7.9	-5.8	-17.6	-	6.1	145
14	1378397	Normal	intact	mobile	present	33,31	refer	-8.55	-6.5	-6	-13.6	-	9.8	119
15	1326134	Normal	intact	mobile	present	56,55	refer	-17.3	-13.7	-15	-21.3	-	5.3	134
16	1288357	Normal	intact	mobile	present	40,56	refer	-19.8	-14.1	-16	-23.9	-	6.6	149
17	1415677	Normal	intact	mobile	present	21,21	refer	-15.2	-11.8	-10	-14.1	-	7.7	162
18	1318533	Normal	intact	mobile	present	86,100	refer	-11.5	-8.1	-9.2	-12.1	-	6	129
19	635597	Normal	intact	mobile	present	45,56	refer	-3.9	-2.6	-1.6	-10.4	-	10.8	156
20	682654	Normal	intact	mobile	present	21,46	refer	-11.1	-6.6	-8.5	-17.2	-	9.7	171
21	1008789	Normal	intact	mobile	present	23,16	refer	-2.5	-0.9	-0.7	-15.4	-	11.3	174
22	315777	Normal	intact	mobile	present	21,21	refer	-23.7	-15.9	-1.5	-23.7	-	9.1	158
23	1181695	Normal	intact	mobile	present	21,21	refer	-17.5	-13.6	-15	-11.2	-	7.9	163
24	1427529	Normal	intact	mobile	present	43,51	refer	-15.74	-12.4	-17	-18.9	-	7.1	132
25	1408681	Normal	intact	mobile	present	30,26	refer	-3.1	-7.8	-0.9	-10.5	-	8.4	155
26	1415677	Normal	intact	mobile	present	78,21	refer	-2.7	-6.89	-0.8	-11.8	-	8.9	139
27	1380986	Normal	intact	mobile	present	40,50	refer	-8.8	-6.7	-5.2	-12.4	-	6	146
28	431301	Normal	intact	mobile	present	20,18	refer	-12.7	-8.33	-4.3	-18.7	-	6.6	163
29	49133	Normal	intact	mobile	present	11,11	refer	-16.4	-12.4	2.3	-17.6	-	5	178
30	654439	Normal	intact	mobile	present	23,45	refer	-10.3	-7.47	-10	-7.86	-	7	154

Baba Farid University of Health Sciences
Sadiq Road
Faridkot-151203 (PB) INDIA
Phone: 01639-250211, 250730
Tele Fax: 01639-250745
Email: Examinations@bfuhs.ac.in

No. BFUHS/2k21p/TH/ 1252 Date 27/10/21

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.10.2021 has approved the plan of thesis of **Hardeep Kaur**, a student of your college for **MS Otorhinolaryngology (ENT)** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Hearing loss in patients with dm-2 (adult) to detect site of lesion by oae and bera.

Supervisor
Dr. Manish Munjal
Prof. & Head,
Dept. of E.N.T.,
DMCH, Ludhiana.

Co-Supervisor

Dr. Sanjeev Puri
Asst. Prof.,
Dept. of E.N.T.,
DMCH, Ludhiana.

Dr. Naveen Mittal
Prof.,
Dept. of Endocrinology,
DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

151. If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.

152. The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.

HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN

PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MASTER OF SURGERY

IN

OTORHINOLARYNGOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

MARCH, 2021

DR HARDEEP KAUR

DEPARTMENT OF OTORHINOLARYNGOLOGY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	HEARING LOSS IN PATIENTS WITH DM-2 (ADULT)TO DETECT SITE OF LESION BY OAE AND BERA
For the degree of	M.S. ENT
Name of the candidate	Dr. HARDEEP KAUR
Supervisor	Dr. Manish Munjal, Professor & Head, Department of ENT, DMCH, Ludhiana
Co-Supervisor	Dr. Sanjeev Puri, Assistant Professor, Department of ENT, DMCH, Ludhiana Dr Naveen Mittal, Professor, Department of Endocrinology, DMCH, Ludhiana
Institution	Dayanand Medical College and Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: OAEs are the low intensity sound produced by the outer hair cells of normal cochlea. It is simple noninvasive screening tool used to assess the function of cochlea. BERA is a non invasive test used to diagnose retro cochlear pathology.

Aims and Objectives: Primary Aim -To detect site of lesion (cochlear hair cell/ cochlear nerve. Secondary Aim -Correlation between diabetic profile (Blood Glucose, Serum HbA1c) and audiological parameter (PTA, OAE, BERA).

Materials and Methods: In this prospective duration based clinical study Diabetic patient with hearing loss will undergo PTA testing if PTA result comes out with finding of SNHL then further investigations such as OAE and BERA will be done to rule out the site of lesion..

Clinical significance: Screening and Early detection of hearing loss in Diabetic patients and the timely management of the same by using OAEs and BERA as a screening tool.

Keywords: Otoacoustic emissions, BERA, Diabetes, Sensorineural hearing loss

(Signature) Candidate | (Signature) Co-supervisor | (Signature) Co-supervisor | (Signature) Supervisor | (Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA"** exist in the Department of ENT at Dayanand Medical College and Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

Dr. Manish Munjal
Professor & Head
Department of ENT
DMCH, Ludhiana

Co-Supervisor

Dr. Sanjeev Puri
Assistant Professor
Department of ENT
DMCH, Ludhiana

Dr. Naveen Mittal
Professor
Department of Endocrinology
DMCH, Ludhiana

Head of the Department

Dr. Manish Munjal
Professor & Head
Department of ENT
DMCH, Ludhiana

Place : _____

Date : _____

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Hardeep Kaur
Department	ENT
Topic of Thesis	HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA
Likely date of appearing in PG examination	MAY 2023
Date of enrollment	JULY 26, 2020
Name of Head of Department	Dr. Manish Munjal
1) Supervisor **2) Co-Supervisor(s)**	Dr. Manish Munjal Dr. Sanjeev Puri Dr. Naveen Mittal
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)	Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

Dr. Sandeep Puri
Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	DR. HARDEEP KAUR FATEHPUR KHURD, TEHSIL GARHSHANKAR, DISTT. HOSHIARPUR
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL, LUDHIANA
3	COURSE OF STUDY AND SUBJECT	M.S.ENT
4	DATE OF ADMISSION OF COURSE	26TH JULY 2020
5	TITLE OF THE TOPIC	HEARING LOSS IN PATIENTS WITH DM-2 (ADULTS) TO DETECT SITE OF LESION BY OAE AND BERA

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Otoacoustic emissions (OAE) is the low intensity sound produced by the inner ear. These emissions will be disturbed when cochlea is damaged. OAE will be normal if there is retro cochlear pathology but there will be abnormal auditory brain stem responses [1,2]. Epidemiology - According to estimation 40.9 millions people in India are suffering from Diabetes Mellitus and by 2025 the number would be around 69.92 million [3,4]. Diabetes is a chronic health condition that causes considerable morbidity and mortality and affects the quality of life[5]. The organ of corti is the sense organ for hearing and the potential target for hyperglycemic damage that occurs in Diabetes Mellitus. Hearing loss affects the quality of life and leads to depression, anxiety as well as social isolation [6]. Diabetes Mellitus and Hearing loss (SNHL) in early studies conducted showed a weak correlation[7,8] but some studies showed positive correlations[9]. Hearing tests evaluates frequencies of 500, 1000, 2000, 3000, 4000, and 6000 Hz. The average of both ears is used for analysis. The pure tone audiometry of both ears at each frequency is obtained for frequency analysis. An average hearing threshold _> 26 db was defined as hearing loss and degree of hearing loss is categorised as mild 26~40 dB, moderate 41~55 dB, moderately severe 56~70 dB and severe 72~ 90dB, profound >91 dB, Deafness (i.e.no hearing) is based on standard of ISO [10]. Microangiopathy in Diabetes Mellitus leads to sensorineural haring loss in diabetic patients [11]. Due to hyperglycemia deposition of glycoproteins in small blood vessels occurs that impacts the neurological function in Diabetes patient [12,13]. Amplitude and intensity of the recorded waves for otoacoustic emission is significantly lower in diabetics than controls[14]. Quality of life improves in diabetic patients with the treatment of hearing loss and it is the chronic complication of diabetes mellitus [15]. Cochlea is damaged by the hyperglycemia that occurs in type two diabetes mellitus Due to damage of outer hair cells in cochlea hearing loss occurs in type 2 diabetes mellitus. The number

of undiagnosed cases of undiagnosed diabetes across the globe is approximately 175 million currently. Cochlear hair cells status is assessed by the otoacoustic emission test that is simple, non invasive and efficient indicator. On auditory stimulation sound is produced by the hair cells in the cochlea known as otoacoustic emissions and OAEs sound is picked up by the microphone fitted in the ear canal. Electrophysiological activity of the auditory pathway is evaluated by the Brainstem auditory evoked responses. Integrity of the auditory pathway and early impairement of acoustic nerve is assessed by the this non invasive test [16].

6.2 REVIEW OF LITERATURE

The outer hair cells in inner ear produce soft sounds (OAEs). These hair cells are electro motile. The main function is the active process that increase sensitivity to sound known as cochlear amplification. This sensitivity to sounds decreases due to damage and loss of outer hair cells. Sometimes without any obvious damage to outer hair cells endo cochlear potential decrease. There are two types of OAEs that are utilized-

(a) Spontaneous OAEs –

They are present in healthy normal hearing persons where hearing loss doesn't exceed 30 dB. They may be absent in 50% of normal persons.

(b) Evoked OAEs –

They are further divided into two types depending on the sound of stimulus used to elicit them.

(1)- Transient evoked OAEs (TEOAEs) –

Evoked by clicks .A series of click stimuli are presented at 80- 85 dB SPL (sound pressure level) and response recorded.

(2)- Distortion product OAEs (DOAEs)-

Two pure tones are simultaneously presented to cochlea to produce distortion product. They have been used to test hearing in range of 1000- 8000Hz.

USES AND APPLICATIONS

1) OAEs are used for screening test for hearing in presbycusis and to test mentally and uncooperative individuals after sedation.

2) They help to distinguish cochlear from retro cochlear pathology. OAEs are also useful to diagnose retrocochlear pathology, especially auditory neuropathy incombination with BERA thus we can clearly distinguish between sensory and neural pathologies.

3) OAEs assess the function of OHCs of organ of corti which is the predominant site of damage In ototoxocity.

4) Monitoring cochlear function during recovery from cochlear dysfunction.

5) OAEs can be either present or absent with 8th nerve tumours, depending on whether the cochlear blood supply has been compromised. If present, OAEs indicate possibility of hearing recovery with a conservative surgical approach.

6) OAEs are objective and require minimal patient cooperation hence can be used in malingers.

7) This test is frequency specific and relatively consistent and stable.

8) The test is short and need not to be performed in a sound attenuated room, though a quiet ambience is essential.

Sound produced by the outer hair cells transmitted by the basilar membrane and perilymph to the oval window then viberates to the ossicles and exits the middle ear by viberating the tympanic membrane and thus reaches the ear canal where it is picked up by the microphone[17].

Hyperglycemia in diabetes causes pathological changes at cellular level .In subjects with type two diabetes mellitus hyperglycemia damage the vessels and nerves at cellular level leads to neuronal degeneration of auditory system. M ORTS et al conducted study on twenty diabetic subjects and the there is significant decrease in mean amplitude of click evoked OAE and DPOAE and comparing the results those obtained in a control of non diabetics[18]. Pemmaiah KD,Srinvas DR conducted study on 110 diabetic patients out of which 48 percent(43.6%) had bilateral sensorineural hearing loss(2000 hz,4000 hz).In 7 patients severe hearing loss was seen(6.36%) ,in 16 patients (14.54%)moderately severe hearing loss,in 25 patients moderately severe hearing loss was seen .Among 47 patients who had DM for more than 10 yrs, 29 patients (61.7%) showed at least mild hearing loss .In diabetic patient progression of hearing loss can be modified by good glycaemic control.[19] Vaughan et al conducted study on adult military veteran including (27 women and 667 men)of whom 352 didn't had diabetes and 342 had diabetes, high frequency hearing loss found

in those with diabetes under 60 yrs of age than those without diabetes[20]. Mitchell et al, conducted retrospective analysis of a population survey that followed up the participants for 10 yrs. The researchers used data from 2334 participants year-5 follow up visit and 11952 participants year 10 follow up, as audiometric testing was done at these two times found that hearing loss was worse at all frequencies between 250 and 8000hHz [21]. Frisina et al cite hearing loss in diabetes mellitus occurs due to Reactive oxygen species [22]. Soveid et al conducted study on 78 healthy diabetics and 80 diabetic of mean age 49 years and compared tonic and verbal audiometry. Speech impairement and hearing loss were lower in controls than diabetics [23]. Konrad -Martin et al conducted a study in 138 healthy controls under the age of 50 years and 166 diabetics and investigated ABR in these case control groups, disorderd ABR reported in diabetic group [24]. Amplitude of otoacoustic emission was lower and latency of ABR was greater in diabetic group as compared to non diabetics in study conducted by the Lisowska [25].

6.3 AIMS AND OBJECTIVES OF THE STUDY

- **PRIMARY AIM –** To detect site of lesion (cochlear hair cells / cochlear nerve)
- **SECONDARY AIM-** To find Correlation between Diabetic profile (blood glucose, HB1AC) and Audiological parameters (PTA, OAE, BERA)

7. MATERIALS AND METHODS

7.1 Source of Data

This Prospective study will be conducted in Department of ENT, Dayanand Medical College, for a period of one and a half year. This study group includes diabetes patient with hearing loss. All subjects after informed consent will undergo standard audiometry, OAE and BERA testing to detect the site of lesion.

7.2 METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA

Inclusion criteria would be

1) Adult diabetic patients with hearing impairment.
2) Patients Consenting for the study.

EXCLUSION CRITERIA

1) Patients with hearing loss secondary to trauma (Head injury) with documented temporal bone fracture.
2) Presence of any external or middle ear pathology (e.g. stenosis of EAC , otitis media, tympanic membrane perforation, cyst, otosclerosis).
3) Family history of sensory neural hearing loss.
4) Noise exposure.
5) Patient with Ear infections
6) Neoplastic lesion of temporal bone .
7) Patient who not consenting for the study.

CONSENT – An informed consent will be obtained from the patient explaining the need of monitoring in diabetic patients with hearing loss.

INVESTIGATIONS-

PTA

OAE TEST

BERA

SERUM HBA1C

BLOOD GLUCOSE TEST

STUDY DURATION – ONE AND HALF YEARS from 1st March 2021 to 31st Aug. 2022

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

1) Medical History - A detailed history will be taken to enquire diabetic patients with complaints of hearing loss and will be analysed prospectively regarding Duration of the disease, blood glucose levels, serum HbA1c, Treatment modalities (OHA, insulin).

2) Clinical Examination –

ENT examination- Examination of the Ear, external appearance Otoscopic examination – EAC, TM, PTA.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith

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PROFORMA

Sr No.

Name of the patient..... Age..... Sex.....

MRD. No.....Weight.....

Address.....

.....

.....

HISTORY

1) H/O Diabetes

2) H/O decreased hearing

3) H/O ear discharge /earache/tinnitus....

4) H/O intake of any ototoxic drug.....

5) H/O head injury with documented temporal bone fracture....

6) H/O exposure to abnormally loud sounds....

7) Family history of SNHL.....

8) Any other mentionable findings....

Signature of Supervisor

Signature of Candidate

EXAMINATION OF THE EAR

External appearance.....

Otoscopic examination....

TM.....

Mobility....

COL.....

BASE LINE INVESTIGATION

1) PTA
2) OAE
3) BERA
4) BLOOD GLUCOSE
5) HBA1C

SR NO.	Otoacoustic Emissions	Pass	Refer

Signature of Supervisor

Signature of Candidate

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....
am willing to participate in the study **"HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

Name:

.....
Signature of the patient

Signature of Candidate

Signature of Supervisor

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"HEARING LOSS IN PATIENTS WITH DM-2 (ADULT) TO DETECT SITE OF LESION BY OAE AND BERA" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ। ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ